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THESIS ABSTRACT

THE OHIO STATE UNIVERSITY
GRADUATE SCHOOL

NAME: Nusstein, John Michael, Major, USAF **QUARTER/YEAR:** Summer 1995

DEPARTMENT: Dentistry, Advanced Endodontics **DEGREE:** Master of Science

ADVISER'S NAME: Reader, Alfred W.

TITLE OF THESIS: The Anesthetic Efficacy of the Intraosseous Injection in Irreversible Pulpitis

The purpose of this study was to evaluate the anesthetic efficacy of an intraosseous injection in teeth diagnosed with irreversible pulpitis. Fifty-one healthy human subjects with symptomatic maxillary or mandibular posterior teeth diagnosed with irreversible pulpitis were used in this study. The subjects were tested with an electric pulp tester and Green Endo-Ice® prior to and after receiving anesthesia to determine pulpal vitality and anesthesia. Teeth which responded to either test after a set time period or patients who felt pain during specific portions of root canal therapy received an intraosseous injection (2% lidocaine with 1:100,000 epinephrine). Subjects were asked to rate any pain experienced during injections, testing, and root canal therapy.

Forty-two percent of patients who tested negative with the electric pulp tester and Green Endo-Ice®, after successful clinical injections, reported pain during treatment. Eighty-one percent of mandibular teeth and 12% of maxillary teeth required an intraosseous injection due to failure in gaining pulpal anesthesia. The Stabident Intraosseous injection was found to be 90% successful in mandibular teeth in gaining total pulpal anesthesia for endodontic therapy. The overall success rate of the Stabident injection was 87.5%.

Adviser's Signature

THE ANESTHETIC EFFICACY OF THE INTRAOSSEOUS INJECTION IN IRREVERSIBLE PULPITIS

A THESIS

Presented in Partial Fulfillment of the Requirements for the Degree of Master of Science in the Graduate School of The Ohio State University

by

JOHN M. NUSSTEIN, D.D.S.

*****

The Ohio State University
1995

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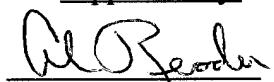

Adviser
College of Dentistry

DEDICATION

To the memory of my father, Johann Nusstein, who taught me the values of hard work, diligence, patience, and attention to detail. All of these values have culminated into a piece of work he would have been very proud of.

ACKNOWLEDGMENTS

I would like to express my gratitude to the following
people for making this thesis possible.

My wife Tammie and son Aaron - None of this work would have gotten completed without your love and support. Now it is off to Japan and our next adventure.

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Dr. William Meyers - Its a sad honor to be in your last graduating class, but I am glad to have had the chance to be under your mentorship and touch wisdom that only experience provides.

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Dr. Mike Beck - Thank you for not overpowering me with statistics and making a difficult project go a lot easier.

My classmates, Dr. Jon Reitz, Dr. Mark Dinkins, Dr. Mark Henry - The job does not get done unless the team works well together and we got the job done very well.

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Adviser's Signature

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CHAPTER I

INTRODUCTION

The main method of controlling pain in dentistry is by the use of local anesthesia. However, achieving profound anesthesia is not always successful (1,2). Kaufman et al. (3) reported, in a survey of general dentists, that problems with anesthesia occurred in 13% of the cases and that the greatest percentage of failure (88%) occurred with inferior alveolar nerve blocks. Weinstein et al. (4) reported the results of a patient survey in which close to 1 in 7 patients was hurt during treatment after receiving anesthesia. Attempts at modifying the basic inferior alveolar injection in the hope of increasing anesthetic efficacy in mandibular teeth have met with little success (5,6,7,8).

Determining the success of anesthesia has always been based on the dentist's evaluation of a patient's reaction during treatment. This tends to be very subjective and dependent on a number of variables (9). The use of the electric pulp tester provides a more objective method of determining anesthesia. Bjorn (10) felt that the electric pulp tester had more precision of control, was easier to use, and simulated physiologic stimuli better than other tests. Dreven (11) found that, using an Analytic Technology® electric pulp tester, a reading of 80/80 meant complete pulpal anesthesia in normal and asymptomatic restored teeth. However, it did not guarantee clinically successful pulpal

anesthesia in teeth with irreversibly inflamed pulps. McDaniel et al. (12) determined that the use of the electric pulp tester, even for up to 5 hours, caused no pulpal damage. The use of cold stimuli may often mimic and cause the same symptoms a patient has been experiencing. It can also help determine pulpal vitality. Numerous authors (13,14,15) have cited the use of cold, either in the form of ice, CO_2 (dry ice), or ethyl chloride for determining pulpal vitality. Fuss et al. (16) however found that the use of dichlorodifluoromethane (DDM) and CO_2 was more reliable than ice or ethyl chloride. It was suggested that DDM may be equal to the use of the electric pulp tester in determining vitality. Cohen et al. (17) found, in teeth with the clinical diagnosis of irreversible pulpitis, that DDM was an excellent, simple, and highly reliable method of testing pulpal anesthesia.

The study of the intraosseous injection had been previously limited to the study of the periodontal ligament injection (18,19,20,21,22,23,24,25). With the development of the Stabident intraosseous injection system, the study of the intraosseous injection can be conducted by the injection of anesthetic solution directly into the cancellous bone (26,27,28).

The Stabident intraosseous injection system uses a handpiece driven perforator, a 27-gauge, solid, beveled wire that makes a hole in the cortical plate of bone. A corresponding 27-gauge ultra-short needle, which is part of the system, is then placed into the perforation to aid in delivering the anesthetic solution directly into the cancellous bone surrounding the tooth to anesthetized.

Dunbar (26) has shown that the use of the Stabident system as a supplemental injection to an inferior alveolar nerve block was 90-100% effective in achieving anesthesia in clinically normal mandibular first molars. He also found that the injection was relatively painless to give. Onset was reported to be immediate and Dunbar also reported a decrease in the percentage of patients having slow onset of anesthesia. Furthermore, he found that the duration of pulpal anesthesia in the mandibular first molar, after a supplemental injection of 1.8 cc of 2% lidocaine with 1:100,000 epinephrine via the Stabident injection, was 90% after 60 minutes.

Coggins (27) found that the success of the Stabident intraosseous injection, given as a primary injection, to be 90% in maxillary lateral incisors; 93% in maxillary first molars; 75% in mandibular first molars; and 78% in mandibular lateral incisors. In terms of pain of injection, he found that the infiltration injection into the gingiva where the perforation was to be done was significantly more painful than the actual perforation of the cortical bone, insertion of the needle, and deposition of the anesthetic solution into the cancellous bone. As a primary injection, he reported that pulpal anesthesia declined steadily over an hour. Approximately 65% of the patients were numb at 30 minutes and 40% were numb at 60 minutes. The mandibular lateral incisors, however, declined at a faster rate.

Replogle (28), utilizing an EKG and Dinemapp® monitor to record heart rate and blood pressure, found that approximately 67% of patients had a rise in cardiac rate during or 2 minutes after deposition of 1.8 cc of 2% lidocaine with 1:100,000 epinephrine by way of the Stabident intraosseous injection. The use of 1.8 cc of 3% mepivacaine did not significantly increase cardiac rate. No significant differences were found in terms of

systolic or diastolic blood pressure changes with either solution. The mean heart rate increase was 22 beats/minute from measured pre-injection rates. The cardiac effect usually dissipated within 2 minutes and would be clinically insignificant in healthy individuals.

These studies (26,27,28) were all conducted on asymptomatic, minimally restored teeth. No studies have been done clinically to determine the effectiveness of the Stabident system on achieving pulpal anesthesia in teeth diagnosed as having irreversible pulpitis. Therefore, the purpose of this study was to determine the anesthetic efficacy of the Stabident intraosseous injection technique, when used as a supplemental injection, in teeth with the diagnosis of irreversible pulpitis.

CHAPTER II

LITERATURE REVIEW

Selected portions of the following literature review were derived from previous Theses by Dreven (29), Uhle (30), Dunbar (27), and Coggins (28) in the Department of Endodontics at The Ohio State University.

MECHANISM OF ACTION OF LOCAL ANESTHETICS

Local anesthetics are agents that reversibly block nerve conduction or depress excitation of nerve endings when applied to a circumscribed area of the body (31). They inhibit the propagation of nerve impulses along fibers, at sensory endings, at myoneural junctions, and synapses (32). Local anesthetics affect small, unmyelinated, or thinly myelinated fibers first and affect large or heavily myelinated fibers last. Therefore, the losses in the usual order (individuals may vary) are autonomics; sense of cold, warmth, pain, touch, pressure, and vibration; proprioception; and motor function (33).

The permeability of the nerve cell membrane, in conjunction with the cytoplasmic and extracellular electrolytic concentrations, determine the electrophysiologic properties of the nerve cell membrane (31). In its resting state, the nerve membrane is slightly permeable to sodium ions, proteins and amino acids while potassium and chloride ions are freely

permeable. (31,34). Potassium remains within the cell's axoplam despite its free permeability to diffuse through the nerve membrane and despite its concentration gradient. This is due to the negative charge of the nerve membrane restraining the positively charged potassium ions by electrostatic attraction (34).

Chloride ions remain outside the nerve membrane, instead of moving across the concentration gradient, because of the nearly equal opposing electrostatic influence mentioned above (34). Also, since the sodium ions cannot readily cross into the cell, a -90 mV electrical potential is maintained across the nerve membrane (31).

Excitation of the nerve causes an increase in the permeability of the cell membrane to sodium ions through a transient widening of the transmembranous channels. The rapid influx of the sodium ions results in a depolarization of the cell membrane to a firing threshold of -50 to -60 mV. Once the nerve fires, a dramatic increase in sodium permeability is noted with a concomitant increase in sodium ion movement across the membrane. At the end of this cycle (depolarization phase) the electrical potential has actually increased to a positive potential of +40 mV (31,34).

After the depolarization phase, nerve membrane permeability to sodium decreases, and the high permeability of potassium is restored. The resting potential is restored by the movement of potassium back into the cell and the "pumping" of sodium out. The "sodium pump" uses energy since it is working against the concentration gradient. The energy for this mechanism is derived from the oxidative metabolism of adenosine triphosphate (31,34). This repolarization phase requires about 0.7 msec. and following this time period the nerve cell regains its original resting-state electric potential of -90 mV (34).

A number of theories have been proposed as to how local anesthetics work on the nerve fiber. The prevention of depolarization by occluding the transmembranous sodium channels is the current accepted theory (31,32,34,35,36,37,38). This occurs by either the specific receptor mechanism and/or the membrane expansion mechanism.

The specific receptor theory describes four binding sites where a local anesthetic molecule may attach. The first site, just outside the inner opening of the channel, results in a tonic block. Binding to the receptor within the channel produces a use-dependent block (36). The other two sites are at the gate of the channel and are related to scorpion venom (32). It is the uncharged form of a local anesthetic that can penetrate a nerve membrane. After penetration, an equilibrium occurs between the charged cationic form and the uncharged base form of the anesthetic. Since the intracellular pH is about 7.35, 75% of the drug converts to the cationic form. It is these charged molecules that bind to the receptor sites and prevent the diffusion of the sodium into the cell and thus prevent firing of the membrane (31,34,37).

The membrane expansion theory states that local anesthetic molecules diffuse to hydrophobic regions of the nerve membrane and cause expansion of these areas. This leads to a decrease in sodium channel diameter and prevents sodium permeability (31,34,38). This theory possibly explains the local anesthetic effectiveness of agents like benzocaine which do not exist in ionized form yet still exhibit anesthetic activity (34).

PHARMACOLOGY OF LOCAL ANESTHETICS

Local anesthetic agents can reversibly alter or modify the feeling in a peripheral area to which they are applied (39). Chemically, dental local anesthetics are made from the hydrochloride salt of the local anesthetic base (40). The local anesthetic molecule can be divided into three parts: an aromatic group, an intermediate chain, and a secondary or tertiary amino terminus (31,35). The aromatic residue of benzoic acid or aniline confers lipophilic properties on the molecule, whereas the amino group furnishes water solubility (35). The intermediate carbon chain helps provide the necessary spatial separation between the lipophilic and hydrophilic ends and it works with the aromatic moiety to help classify the agents as either esters or amides (31,35,41). The differences in the two compounds resides in the manner in which they are metabolized and their allergic potential (31,35).

The ester anesthetics are hydrolyzed in the plasma by pseudocholinesterase (31,35,42). Para-aminobenzoic acid is one of the metabolites formed from the hydrolysis of ester-type compounds. This compound is capable of inducing allergic-type reactions in a small percentage of the general population (31,34).

Metabolism of amide drugs occurs primarily in the microsomes of the liver. The initial reaction is usually N-dealkylation of the tertiary amino terminus (35,39). The resultant secondary amine is susceptible to hydrolysis by hepatic amidase activity (35). The amide anesthetics are not metabolized to para-aminobenzoic acid and thus allergic reactions are rare (31). Excretion of both the amide and ester anesthetics are from the kidney (34,35,41).

The anesthetic profile of a chemical compound is based on its (a) lipid solubility, (b) protein-binding, (c) pK_a, (d) non-nervous tissue diffusibility, and (e) intrinsic vasodilator activity (31).

Lipid solubility appears to be the most important factor in determining the intrinsic anesthetic potency of an agent. As lipid solubility increases, the ability of the anesthetic to penetrate the nerve cell membrane also increases, thus decreasing the anesthetic agent's concentration requirement (31,34,35). Lidocaine, prilocaine, and mepivacaine have been classified as having moderate lipid solubility. Bupivacaine and etidocaine have much higher solubility and are considered more potent (31).

The protein-binding characteristics of an anesthetic agent influences its duration of action. This is because the nerve membrane is 10% protein and agents that can penetrate the membrane and attach more firmly to it will have a prolonged action (31). Poorly binding agents, like procaine, have a relatively short duration of action. Again, lidocaine, mepivacaine, and prilocaine are moderately protein bound while bupivacaine and etidocaine are highly bound and are considered long acting anesthetics. (31,32).

The pK_a of a chemical compound may be defined as the pH at which its ionized and nonionized forms are in equilibrium. Most local anesthetics are weak bases by virtue of the substituted amino group, and have pK_as ranging from 7.5-9.0 (31,34,35,41). At a tissue pH of 7.4, most anesthetics will be from 2% to 40% in nonionized (base) form (31). It is this form of the anesthetic agent that penetrates the nerve cell membrane. Hence, the pK_a of the solution and pH of the tissue will affect onset time. Anesthetic agents whose pK_as come closer to the pH of the tissue will have a more rapid onset (31,32,43).

Prilocaine, etidocaine, and lidocaine, with pK_as of 7.7, have faster onset times than bupivicaine and procaine whose pK_as are 8.1 and 8.9, respectively (31,40).

In non-nervous tissue, anesthetics have variable rates of diffusion. While the onset of local anesthesia is related to the diffusion properties of the agent used, the factors that determine diffusibility through non-nervous tissue are unclear (31).

The intrinsic vasodilator activity of different local anesthetic agents will influence their apparent potency and duration of action. The vasodilatory effect of the anesthetic agent can increase the blood flow through the area where the drug is deposited. This can lead to faster removal of the local anesthetic from the site and can adversely affect duration and potency of the agent (31).

VASOCONSTRICTORS

Vasoconstrictors are agents that initiate narrowing of the lumen of blood vessels and thereby control tissue perfusion. Most vasoconstrictors used in local anesthetic solutions are classified as adrenergic or sympathomimetic amines because they are closely related to the natural mediators of the sympathetic nervous system. Because clinically useful local anesthetics are all vasodilators to some degree, their injection into tissue will increase blood flow, and this leads to a decrease in duration and effectiveness of the agent. Vasoconstrictors are added to enhance the duration and effectiveness of anesthesia, decrease systemic toxicity by lowering blood concentrations of anesthetic, and decrease bleeding at the injection site (31,32,34,35,41,43,44,45).

The most commonly used vasoconstrictors in dental anesthetics are epinephrine and levonordefrin (43). Epinephrine acts directly on both alpha and beta adrenergic receptors. However, the beta effects predominate. Levonordefrin appears to act through direct alpha receptor stimulation with little or no beta activity, but its vasopressor potency is only about one sixth of epinephrine's (34,43,44). Activation of alpha receptors produces a response that includes contraction of smooth muscle in blood vessels (vasoconstriction). Activation of beta receptors produces smooth muscles relaxation and cardiac stimulation (34,44,45).

Following an intraoral submucosal injection of a local anesthetic containing epinephrine, the alpha effects predominate locally and vasoconstriction occurs. Regional blood flow is reduced and this enhances the duration and efficacy of the anesthetic. It also decreases systemic uptake and causes hemostasis. The effects may last 30 to 90 minutes

following injection. Gradually, the local tissue concentration of epinephrine decreases to a level that no longer produces an alpha-adrenergic effect and the beta response predominates. Local blood flow increases and the hemostatic effect of the epinephrine is lost (43).

Vasoconstrictors, at the dosages used in local anesthetics in dentistry, have no clinical effect other than the direct action on the alpha receptors (34). Patients with hypertension, cardiovascular disease, and hyperthyroid may be sensitive to the pressor responses of the vasoconstrictor and, therefore, these agents should be used carefully (34,35,44).

The use of vasoconstrictors and the administration of certain medications may be a contraindication for use. Drug interactions with monoamine oxidase inhibitors, tricyclic antidepressants, beta-blockers, and phenothiazines have been documented (32,43,46,47,48). However, in a recent study by Yagiela et al. (47), they demonstrated that epinephrine, levonordefrin, and norepinephrine may be used in patients taking monoamine oxidase inhibitors or phenothiazines. Vasoconstrictors may also be used in patients taking tricyclic antidepressants, but the dosage should be kept to a minimum (0.05 mg, or 3 cartridges of 1:100,000 epinephrine). Absolute contraindications to epinephrine use include pheochromocytoma and thyrotoxicosis (43,49).

CARDIOVASCULAR EFFECTS OF LOCAL ANESTHETICS AND EPINEPHRINE

Several studies have considered the cardiovascular effects of local anesthetics and epinephrine in various concentrations. Knoll-Kohler et al. (50) studied the effects on cardiovascular hemodynamics of maxillary infiltrations using 2% lidocaine with and without 1:100,000 epinephrine. In their study, injections using 2% lidocaine plain produced no significant changes in heart rate when compared to baseline values. The injection of 2% lidocaine with 1:100,000 epinephrine caused a slight increase in heart rate. In addition, no correlation was found between heart rate increase and the measured concentrations of epinephrine in venous plasma. Similarly, cardiovascular effects were compared with plasma levels of epinephrine by Tolas et al. (51) following maxillary infiltration injections. Arterial plasma epinephrine concentration and cardiovascular hemodynamic variables remained unchanged after injection of lidocaine. However, a two to three fold increase in plasma epinephrine, a small decrease in mean arterial blood pressure, and a slight increase in heart rate were seen following injection of 2% lidocaine with 1:100,000 epinephrine. The changes in the heart rate and blood pressure were found to be statistically insignificant.

Salomen et al. (52) compared 2% lidocaine with and without 1:80,000 epinephrine using the inferior alveolar nerve block. While the injection of lidocaine without epinephrine caused no change in the heart rate, blood pressure or plasma concentration of epinephrine, injection of lidocaine with epinephrine resulted in an increase in heart rate, ten fold increase in plasma levels of epinephrine, but no change in blood pressure. They

showed no statistical significance in the correations between plasma levels of epinephrine and the hemodynamic data. Additionally, they stated that the slight increase in heart rate would not be clinically significant in healthy individuals. The effects of 2% lidocaine with 1:80,000 epinephrine on heart rate and blood pressure in correlation with plasma potassium levels was studied by Meechan and Rawlins (53). The cardiovascular changes were found to be negligible. The decrease in plasma concentration of potassium, due to the increase in plasma epinephrine, was significant, but not correlated to any cardiovascular changes.

With patients being treated for cardiac disease, Vanderheyden et al. (54) evaluated the effects of both a maxillary infiltration and an inferior alveolar nerve block with 2% lidocaine with 1:100,000 epinephrine on heart rate, blood pressure, and myocardial ischemia. The results showed a slight increase in heart rate, systolic blood pressure, and rate pressure product (heart rate x systolic blood pressure), but changes were not found to be significant and myocardial ischemia was not provoked by the lidocaine with 1:100,000 epinephrine.

Cardiovascular responses to intraosseous injections of lidocaine with and without epinephrine were studied by Lilienthal and Reynolds (55). They compared 0.9 ml of 2% lidocaine without epinephrine and 0.9 ml of 2% lidocaine with 1:80,000 epinephrine in infiltration and intraosseous injections. No changes were seen in heart rate nor blood pressure using the 2% lidocaine plain in either injection type. The 2% lidocaine with 1:80,000 epinephrine did not cause any change in cardiovascular dynamics with the infiltration injection, but it did cause a rapid elevation of heart rate and blood pressure

when given intraosseously. The values returned to normal within 2 to 3 minutes following the intraosseous injection. In another study, Lilienthal (56) used 1.0 ml of 4% prilocaine with and without 1:200,000 epinephrine given by intraosseous injection to look at the cardiovascular responses. Even with the decreased concentration of epinephrine there was a significant rapid increase in heart rate and a slight increase in blood pressure. Again, the heart rate and blood pressure returned to normal within 2 to 3 minutes. Prilocaine without epinephrine had no effect on the heart rate or blood pressure. Lilienthal recommends, on the basis of his results, that the intraosseous injection of local anesthetics containing epinephrine be restricted to healthy patients.

Cannell and Cannon (57) compared the circulating levels of 2% lignocaine with and without 1:80,000 epinephrine using an intraosseous injection. They reported that their subjects, those receiving the lignocaine with 1:80,000 epinephrine, experienced faintness, pallor, feelings of anxiety, palpitations, and tremor. None of these symptoms were reported after an intraosseous injection of lignocaine without epinephrine. The circulating levels of both agents were comparable with direct intravenous injections, and the presence of epinephrine had no limiting effect on the circulating levels of the lignocaine.

Coggins (27), Dunbar (26), and Replogle (28) found, using the Stabident intraoseous injection system and 1.8 cc of 2% lidocaine with 1:100,000 epinephrine, that approximately 75% of patients reported a subjective feeling of inceased heart rate. Replogle (28) also used 3% mepivacaine plain and found that none of the patients reported a feeling of increased heart rate.

Replogle (28) used an EKG machine and a Dinemapp® monitor to record heart rate and blood pressure during and after an intraosseous injection using the Stabident system with either 2% lidocaine with 1:100,000 epinephrine or 3% mepivacaine. She found heart rates to increase significantly during and 2 minutes after the injection of the 2% lidocaine with 1:100,000 epinephrine as compared to the 3% mepivacaine. No differences were found between the solutions regarding either systolic or diastolic blood pressure. Approximately 67% of the patients had a recorded rise in cardiac rate during or 2 minutes after deposition of the lidocaine solution. The mean rise in heart rate was 22 beats per minute above the pre-test rate. This was reported as not clinically significant in healthy patients (28).

ANATOMICAL CONSIDERATIONS

The Maxillary Nerve

The trigeminal nerve is the largest cranial nerve. It provides the overwhelming majority of sensory innervation to the teeth, bone, and the soft tissues of the oral cavity (34).

The sensory root fibers of the trigeminal nerve are located in Meckel's cavity on the anterior surface of the petrosal portion of the temporal bone. The peripheral processes of the trigeminal ganglion constitute the ophthalmic and maxillary nerves and the sensory component of the mandibular nerve (34,58,59). The maxillary nerve arises from the middle of the trigeminal ganglion and passes through the cavernous sinus, exiting the cranium through the foramen rotundum. As the nerve crosses the pterygopalatine fossa, branches are given off to the sphenopalatine ganglion, the posterior superior alveolar nerve, and the zygomatic branches (34,58). The nerve then angles laterally in a groove on the posterior surface of the maxilla, entering the orbit through the inferior orbital fissure. Within the orbit it enters the inferior orbital fissure and becomes the infraorbital nerve which then courses through the infraorbital canal. Upon exiting the infraorbital foramen, the nerve divides into its terminal branches which supply the skin of the face, nose, lower eyelid, and upper lip (34,58). Within the infraorbital canal the maxillary division gives off the posterior, middle and anterior superior alveolar nerves (34,58,60). The function of these nerves is to provide pulpal innervation to the maxillary teeth as well as sensory innervation to the associated buccal mucosa, periodontal tissues, and buccal bone (34,58). The infraorbital nerve gives rise to the posterior superior alveolar nerve just prior to it

entering the infraorbital groove (60). The posterior superior alveolar nerve may be represented by one or more branches. The nerve passes through the pterygopalatine fossa and enters the posterior alveolar canal along with the maxillary artery (58,60). A branch of the nerve may not enter the bone but run laterally to innervate the adjacent facial mucosa and buccal gingiva in the maxillary molar region (58,60). The pulpal tissues and supporting structures of the maxillary second and third molars and the palatal and distobuccal roots of the first maxillary molars receive sensory innervation from the posterior superior nerve (34).

Loetscher and Walton (61) dissected 29 human maxillae to determine the source of pulpal innervation in the maxillary first molar. The posterior superior alveolar nerve could be traced to the apices of the first molar in 72% of the specimens. When present, the middle superior alveolar nerve contributed innervation 28% of the time.

The middle superior alveolar nerve branches off the main nerve trunk within the infraorbital canal. It can arise as a separate nerve anywhere along the main branch and sometimes as far anterior as the origin of the anterior superior alveolar nerve (34,58). The nerve provides sensory innervation to the two maxillary premolars and the mesiobuccal root of the first molar, as well as their periodontal tissues. Malamed (34) reports that the nerve may actually be absent in 60% to 80% of individuals. In the absence of the middle superior alveolar nerve, the posterior or, more frequently, the anterior superior alveolar nerve will supply its usual innervations (34). Loetscher and Walton (61) reported that in 53% of the maxillae in which the middle superior alveolar nerve was absent, secondary

branches were released from the anterior superior alveolar nerve which descended to the area of the premolars.

The anterior superior alveolar nerve branches off approximately 6-10 mm before the exit of the infraorbital nerve from the infraorbital foramen. It descends within the anterior wall of the maxillary sinus. The anterior superior alveolar nerve provides pulpal innervation to the central and lateral incisors and canine as well as sensory innervation to the periodontal tissues, buccal bone, and mucous membranes of these teeth (34). The nerve may also give off small nasal branches that innervate the anterior part of the nasal cavity, along with branches of the pterygopalatine nerves (34,60).

The innervation of individual roots of all teeth, bone, and periodontal structures in the maxilla derive from terminal branches of larger nerves in the region. This is called the superior dental plexus. It is comprised of smaller nerve fibers from the three superior alveolar nerves (34,58). Three types of nerves emerge from this plexus: (1) dental nerves, (2) interdental branches, and (3) interradicular branches. It is the dental nerves which enter the tooth through the apical foramen of the root (34,58). Haesman (62) reported that a superior alveolar nerve plexus was found in 100% of the specimens studied, but that it was impossible to assign the innervation of the maxillary canines and premolars to any definite nerve source.

The Mandibular Nerve

The largest of the three divisions of the trigeminal nerve is the mandibular. It is the only one of the three nerves that caries both sensory and motor innervation. The maxillary and ophthalmic divisions carry purely sensory nerves (34).

The motor division arises in motor cells located in the medulla oblongata. It joins the sensory fibers of the mandibular nerve just distal to the trigeminal ganglion (34,59). The two roots emerge from the cranium through the foramen ovale where they unite to form the mandibular trunk. The nerve divides again into a smaller anterior and large posterior division about 2 to 3 mm after they have joined (34). Branches also come off the undivided trunk. These include the nervus spinosus and the medial pterygoid nerve. The nervus spinosus supplies the dura mater and mastoid air cells while the medial pterygoid nerve is a motor nerve that innervates the internal pterygoid muscle, tensor veli palatini, and tensor tympani muscles (34).

The anterior division provides motor innervation to the muscles of mastication and sensory innervation to the mucous membrane of the cheek and buccal mucosa of the mandibular molars (34). The nerve sends off more branches as it passes between or winds over the two heads of the lateral pterygoid muscle. These branches include the deep temporal nerves which innervate the temporal muscle and the mesenteric and lateral pterygoid nerves that innervate the masseter and lateral pterygoid muscles respectively (34,58). The buccal nerve now remains and it follows the inferior part of the temporal muscle and emerges under the anterior border of the masseter. At the level of the occlusal plane of the third or second mandibular molar it crosses in front of the anterior border of

the ramus and enters the cheek through the buccinator muscle. This nerve provides sensory fibers for the skin of the cheek, retromolar triangle and buccal gingiva of the mandibular molars and the mucobuccal fold. It does not innervate the buccinator muscle (34,58).

The posterior division of the trunk is primarily sensory with a small motor component. It descends before it branches into the auriculotemporal, lingual, and inferior alveolar nerves (34). The auriculotemporal nerve is strictly sensory and divides into superior and inferior branches as it crosses the neck of the mandible. The superior branch provides innervation to the outer ear, external auditory meatus, the temporomandibular joint, and the posterior skin of the temple. The inferior branch joins the facial nerve in the parotid gland to provide sensory innervation to the posterior part of the cheek (34,58).

The lingual nerve is the second branch of the posterior division of the mandibular nerve. It descends medially past the lateral pterygoid muscle to pass between the medial border of the ramus and the medial pterygoid muscle in the pterygomandibular space. It then proceeds anteriorly across the muscles of the tongue to the deep surfaces of the sublingual gland, where it breaks up into its terminal branches (34,58). The lingual nerve is the sensory nerve to the anterior two thirds of the tongue. It provides both general sensation and gustation for its region. The lingual nerve supplies fibers for the former, the chorda tympani (a branch of the facial nerve) the fibers for the latter (34). The lingual nerve also provides sensory innervation to the mucous membranes of the floor of the mouth and the gingiva on the lingual surface of the mandible (34,58).

The inferior alveolar nerve is the largest branch of the mandibular division. It descends between the two pterygoid muscles in the pterygomandibular space along the medial surface of the mandibular ramus entering the mandible through the mandibular foramen just posterior to the lingula (34,58). It is accompanied on its path by the inferior alveolar artery and the inferior alveolar vein. Prior to its entry into the mandible it gives off a branch called the mylohyoid nerve. This nerve runs in the mylohyoid groove of the mandible and supplies motor innervation to the mylohyoid muscle and the anterior belly of the digastric muscle (34,58). It is thought that this nerve may contain sensory innervation to the mandibular incisors and it has been proposed that it may provide pulpal innervation to portions of the mandibular molars (34,63). Once the inferior alveolar nerve enters the mandible, it travels anteriorly. The posterior and middle dental branches are given off in the canal providing sensory innervation to the pulps and periodontal tissues of the mandibular posterior teeth. As the nerve reaches the premolar area it divides into two terminal branches: the incisive nerve, and the mental nerve. The mental nerve exits from the mandibular canal through the mental foramen and divides into three branches that innervate the skin of the chin and the skin and mucosa of the lower lip, and to the gingiva on the labial surface of the mandible (34,58). The incisive nerve remains within the mandibular canal and forms a nerve plexus that innervates the pulpal tissues of the mandibular canines and incisors via the dental branches (34).

THE ANALYTIC TECHNOLOGY® ELECTRIC PULP TESTER

The Analytic Technology® pulp tester is a device used to assess the vitality of teeth in dentistry. It is powered by 6 volts from four 1.5 volt AA batteries. The unit has a high internal impedance of 150 Kohms, with a voltage range from 15-300 volts. The amperage output ranges from 0 to 50 microamperes. The unit produces electrical stimulus of negative polarity, delivered in bursts of ten pulses separated by a space ten pulses long (11,29,64). The unit is a constant current type electric pulp tester, in that the current will remain stable no matter what resistance is encountered (29,64).

The electric pulp tester has a range of 0-80, with 0 signifying no electrical output and 80 signifying full power output. When the probe of the instrument contacts the tooth, the pulp tester turns on automatically. This feature prevents testing unless a good electrical contact has been established. After electrical contact with the tooth, the intensity of the electrical stimulus will increase automatically. The rate of increase may be adjusted using the rate control dial (29,64). If the full power output of 80 is reached, the red indicator light on the probe flashes. When the probe is removed from the tooth, the digital readout remains fixed at the existing reading. The display will automatically reset and a new testing cycle is initiated when the probe is moved to the next tooth. A delay of 10-15 seconds between tests causes the unit to automatically shut off. The unit also indicates when the battery voltage is low by flashing two red dots on the digital readout (64).

Nerves will be excited where the current density is sufficiently high and in the appropriate direction to depolarize their membranes beyond threshold (65). There are unique problems associated with attempting to excite pulpal nerves. These include the

fact that pulpal nerves require a high current density for excitation (66) and that enamel has a very high resistance to electrical current (67,68). Cooley and Robison (67) estimated the resistance of enamel to be 1-5 million ohms. Mumford and Newton (68) estimated the resistance to be 4.5 million ohms. In order to overcome this problem, the probe of the electric pulp tester must be on tooth structure and the tooth must be dry (69). The use of an electrolyte, such as toothpaste, can help pass the current to the tooth (67,69). A dry tooth also helps prevent current from spreading into peridental structures and forces the majority of the current to travel through the enamel (70).

Dentin has less resistance than enamel. Therefore, the majority of current that comes through the enamel will be channeled through the dentinal tubules (65,68). This high current density is what stimulates pulpal nerves. It has been theorized that the location of nerve stimulation occurs at the pulpodentinal junction (68,70). The size of the nerve can also affect how much current is needed to cause firing (65). A large pulp will have a great number of nerve fibers in the dentinal tubules and at the pulpodentinal junction. If the pulp is small, the nerve will be stimulated in the outer areas of the pulpal core (65).

Kitamura et al. (71) found that the Analytic Technology® pulp tester was 100% clinically accurate for non-vital teeth and 99% clinically accurate for vital teeth. Cooley et al. (72) reported that only two of thirty (6.6%) endodontically treated teeth gave false positive responses. Dreven et al. (11,29) found that a reading of 80/80 correlated with clinical analgesia in normal and asymptomatic teeth and that an electric pulp tester could be used experimentally to accurately evaluate local anesthetic techniques and solutions in normal teeth. They found that 27% of the teeth with irreversible pulpitis did not have

profound anesthesia even after an 80/80 reading was obtained. Del Santo et al. (73) found that the Analytic Technology® digital pulp tester could provide reproducible results when testing hard tissue sites and that no adaptation of the pulp tissue occurred which may affect the levels of current required to excite the pulpal nerves.

COLD THERMAL TESTS

Cold stimuli has been used as a diagnostic tool to determine pulpal vitality in clinical dentistry. It can often mimic and cause the same symptoms the patient has been experiencing. A number of sources of cold stimulation are available. They include ice, CO_2, ethyl chloride, and cold refrigerants.

Dry Ice (CO_2)

The use of dry ice in dentistry was introduced by Back (74) in 1936. Obwegeser and Steinhauser (75) developed an apparatus that was more clinically usable by keeping liquid CO_2 under pressure in a tank which then could be converted into carbon dioxide snow when sprayed into a Plexiglas tube. This could then be used as a CO_2 pencil to test all the teeth in the mouth. Fuhr et al. (76) found that, in vitality testing, CO_2 snow was 96.6% accurate, whereas, electrical and heat tests were 90% and 87.6% accurate, respectively. Fuss et al. (16) also found CO_2 to be more reliable than the electric pulp tester to test vitality in teeth of young patients. In adults, dry ice, the electric pulp tester, and the skin refrigerant dichlorodifluoromethane were all found to be more dependable than ice and ethyl chloride in determining pulpal vitality. The temperature of dry ice has been measured and reported to be -119 °F (77). Augsburger and Peters (78), applying dry ice as it is used clinically to a temperature probe, reported that dry ice produced a low reading of only

–69°F, and that it took almost 20-30 seconds to reach and maintain this temperature.

Possible tooth damage in the use of dry ice was first reported by Lutz et al. (79) and Bachmann et al. (80). They found, using fluorescent UV photography, that the use of dry

ice may create new fissures or cracks in the enamel of teeth and also expand ones already present. Peters et al. (81) did not find any new cracks or fissures after applying carbon dioxide to enamel for 2 minutes and examining the teeth by a profile analyzer or replicas of the enamel surfaces by a scanning electron microscope. Ingram et al. (82) also found this to be true using dog teeth. Peters et al. (83), in an in vivo study, found that carbon dioxide did not cause any changes to the enamel surfaces of 10 teeth even after 2 minutes of exposure.

The effects of cold stimulus on pulp tissue has also been studied. Langeland et al. (84) have shown that lowered pulpal temperatures (to a temperature of +11 °C) caused no pulpal damage, but that extreme cold (-160 °C) for 3 minutes would cause severe tissue damage. Frank et al. (85) showed that, if freezing of the pulpal tissue occurred, pulp degeneration would ensue. Dowden et al. (86) showed, using liquid nitrogen, that even at -80 °C, pulpal damage occurs but the tissue remains vital. Ingram (82) showed that the use of carbon dioxide for up to 2 minutes did not cause any pulpal changes. Augsburger (78) found that, in vitro, a five second application of CO_2 caused a 4-5 °F decrease in pulpal temperature. This compared to a 2-3 °F decrease using the cold refrigerant Fluoro-Methane, and a 1-2 °F decrease using ice.

Ethyl Chloride

Ethyl chloride comes in a spray can and can be applied to a cotton pledget and then held on the test tooth. It reaches a temperature of -10 °C when sprayed on skin. It is more convenient to use than carbon dioxide, but Mumford et al. (87) and Seltzer et al. (88) found that ethyl chloride was unreliable in determining pulp vitality. Fuss et al. (16)

found CO_2, dichlorodifluoromethane, and the electric pulp tester were more reliable in determining pulpal vitality when compared to ethyl chloride and ice.

Cold Refrigerants

The cold refrigerants include dichlorotetrafluoroethane (DTE), fluoro-methane (FM), dichlorodifluoromethane (DDM), and 1,1,1,2 tetrafluoroethane (TFE). All of these compounds come in aerosol cans and they too are sprayed on cotton pledgets and then applied to teeth using cotton forceps. DDM reaches a temperature of -50 °C when sprayed on skin (16) while TFE reaches -26 °C (89). White et al. (90) found that DTE produced a greater thermal change in the pulp chamber, in vitro, as compared to ice sticks or an ice-water bath. Fuss et al. (16) found DDM to be as reliable as dry ice and the electric pulp tester in determining pulpal vitality. Cohen et al. (17) found DDM to be an excellent and reliable method to assess pulpal anesthesia. DDM, FM and DTE are no longer commercially available due to potential environmental hazards. TFE, which has no ozone depletion potential, is sold by The Hygenic Corp. as Green Endo-Ice® (89).

THE DENTAL PULP

The dental pulp is a specialized loose connective tissue system composed of cells, ground substance, and fibers (15). It is similar to other connective tissues with the exception of its location (encased in dentin) and that it contains odontoblasts which form dentin (15).

Morphology

Seltzer and Bender et al. (88) state that there is a wide variance in the histologic appearance of normal pulp tissues. However, there are some general characteristics which are considered normal for all pulps. The outermost layer contains a single layer of odontoblasts arranged in a characteristic palisading morphology (13). Central to the odontoblasts is the subodontoblastic layer which has been termed the cell-free zone of Weil (91). This layer includes the terminal nerve endings and capillary plexuses. The cell-rich zone is the next layer. It contains a great majority of the fibroblasts and undifferentiated mesenchymal cells of the pulp (13,91). The core of the pulp follows. It is made up of loose connective tissue and contains the larger blood vessels, lymphatics, and nerve fibers in a network of collagen fibers (92).

Pulpal Inflammation

Inflammation is a descriptive term for the physiologic response of the body to injury (93). Causes for pulpal inflammation include: caries, filling materials, operative procedures, heat, chemicals, and trauma (13,15,92,93). The inflammatory response can be broken down into either acute or chronic phases. Pulpal inflammation is not static. It

does not progress from one phase to another in any particular order (88). Acute and chronic inflammation can be present in the same pulp at the same time (15,88,92).

Acute inflammation is an exudative process in which vasodilatation and increased vascular permeability allow plasma proteins to enter the tissues (93). Polymorphonuclear leukocytes are the predominant cell-type associated with acute inflammation (93). In the pulp, acute inflammation usually occurs after operative procedures (15,92). Pulpal changes can include a disruption of the odontoblastic layer, edema, dilation of blood vessels, and the exudation of inflammatory cells into the odontoblastic layer (15,92). A pulp that is acutely inflamed will either necrose or attempt repair (13,15,36). If repair occurs, chronic inflammation will be seen co-existing with the repair process (15,88).

Chronic inflammation is characterized by a proliferative response and may follow an episode of acute inflammation or may develop alone (93). Lymphocytes and macrophages are the predominating cell types in this response (15,93). Chronic inflammation occurs, generally, as a response to dental caries. As a carious lesion progresses closer to the pulp, more blood vessels dilate and the number of inflammatory cells in the pulpal tissue increases (15). When the pulp is exposed, the tissue reacts with an acute inflammatory response which is superimposed on the chronic inflammation (15,88).

PULPAL STUDIES OF INFLAMMATION

Immunoglobulins in the Dental Pulp

The presence of humoral immune components in normal and inflamed dental pulps was investigated by Pulver et al. (94). Teeth were extracted and the pulp tissue classified as either normal or inflamed based on histologic findings. The presence of immunoglobulins was then determined by way of direct immunofluorescence. Normal pulps were devoid of immunoglobulin-containing cells and were found to have only trace amounts of serum-derived immunoglobulins. Inflamed pulps did contain immunoglobulin-containing cells, of which approximately 60% were IgG. IgA and IgE were also present in significant amounts. The investigators concluded that immune mechanisms may contribute to the pathologic changes seen in inflamed pulp tissues. Hahn et al. (95) found that 73.6% of acutely inflamed pulps contained IgG and 73.3% of normal pulps contained IgG using an immunodiffusion technique. The amounts of IgG in each group had a large variation and no significant difference could be found between the two groups.

Speer et al. (96) compared inflamed and normal dental pulp tissue for immunoglobulins using an immunodiffusion assay. They too found the levels of IgG and IgA to be higher in inflamed pulpal tissue versus noninflamed tissue. The serum immunoglobulin levels were similar in both groups. They concluded that a local immune response did appear to occur in inflamed dental pulp tissue.

Pekovic et al. (97) reported that T cells and plasma cells were present in inflamed pulps. They felt these findings were evidence of the participation of T cells and plasma cells in cell-mediated pulpal autoallergy reactions and that, in diseased pulps, cytotoxic

and Arthus type immunopathologic reactions occurred. Falkler et al. (98) demonstrated, using an Enzyme-linked Immunosorbent Assay (ELISA), that there was antibody activity in the pulp against the microorganisms implicated in the carious process. They also showed that these immunoglobulins were produced in the pulp and were not due to serum leakage from surrounding tissues.

Pain Mediators and Pulpitis

The dental pulp is one of the richest sources of substance P outside of the central nervous system (15). Substance P is a chemical transmitter in trigeminal nociceptive pathways and has been shown to be released by electrical stimulation of the inferior alveolar nerve (99). It has been suggested that substance P may make pulpal neurons more prone to fire due to alteration of the odontoblastic cell membrane (15).

Cyclic adenosine monophosphate (cAMP) has been identified as a messenger that acts as a trigger for many intracellular and metabolic events (100). Increased cAMP levels are associated with synaptic transmission by neurotransmitter release. The neurotransmitters activate adenyl cyclase which then synthesizes cAMP. Thus, transmitters such as histamine, norepinephrine, and serotonin, elaborated during an inflammatory response are capable of increasing cAMP levels in tissues. Greengard (101) showed that increases in cAMP may reduce the transmission of nerve impulses by hyperpolarization of nerves.

Yip et al. (102) demonstrated the presence of cAMP in normal pulpal tissues. Sproles et al. (103) and Bolanos and Seltzer (104) investigated the correlation of pain and the ratio of cAMP to cGMP in pulpal tissues. No variation in the ratios was seen in normal pulps.

Pulps that were irreversibly inflamed showed a higher level of cGMP versus cAMP. However, the differences were not statistically significant.

The role of prostaglandins in the pathogenesis of pulp and periapical tissues was investigated by Torabinejad and Backland (105). They found that prostaglandins have pro-inflammatory effects and can potentiate pain responses by amplifying the properties of pain producing substances such as bradykinin and histamine. Cohen et al. (106) investigated the concentrations of PGE_2 and PGF_{2alpha} in painful and asymptomatic human dental pulps. They found that the PGE_2 levels were significantly higher for teeth in the asymptomatic (caries or restored but no symptoms) group versus teeth in the normal group. The PGE_2 levels for the irreversible pulpitis teeth were significantly higher than the other two groups. The PGF_{2alpha} level in the irreversible pulpitis group was also significantly higher than the other two groups. This result is consistent with other studies showing the hyperalgesic potential of prostaglandins (93,105).

Khayat et al. (107) demonstrated an increase in calcitonin gene-related peptide (CGRP) containing nerve fibers in pulps and periapical areas of inflamed teeth. Byers et al. (108) reviewed the effects of injury and inflammation on pulpal and periapical nerves. They theorized that increases in CGRP sprouting in inflamed pulps may alter the ability of the nerves to be anesthetized due to cytochemical changes throughout the affected nerve fiber. They further postulated that increased CGRP containing nerve fibers may primarily facilitate inflammatory reactions and tissue healing, with sensory activity being a secondary function.

Correlation of Pain with Pulp Histology

Mitchell and Tarplee (109) attempted to correlate clinical symptoms of pulpitis with pulp histology and found that the extent of inflammation could not be determined by the severity of symptoms or any other diagnostic test employed.

Seltzer et al. (88) investigated whether clinical signs, tests, and symptoms could be correlated to the pathologic status of the pulp. They histologically classified extracted teeth and attempted to correlate this with the clinical signs and symptoms, and test results prior to extraction. They found that the severity of pain was patient dependent and only partially related to the severity of the inflammatory response. They concluded that accurate histological diagnoses could only be made from examination of tissues under the microscope and not from clinical diagnostic tests.

Lundy and Stanley (110) also attempted to correlate pulpal histopathology with clinical symptoms in human teeth. They found that severe clinical responses usually accompanied an acute histological state of the pulp. However, the histopathology of experimentally irritated teeth differed from clinically painful teeth.

Seltzer (111) found that teeth with severe or spontaneous pain generally exhibited severe pathosis of the pulpal tissues.

Mendoza et al. (112,113) investigated the ultrastructural changes of the nerves, vasculature, and connective tissue of apical pulpal tissue in teeth with a clinical diagnosis of irreversible pulpitis. These teeth, as compared to normal control teeth, had moderate to severe changes in all of the tissues studied. However, the exact histological condition of these pulps could not be predicted based on clinical tests or radiographs.

INTRAOSSEOUS ANESTHESIA

The Periodontal Ligament Technique

Oral intraosseous anesthesia has been evaluated primarily through the periodontal ligament injection technique. This technique delivers anesthetic solution to the cancellous bone through the cribiform plate into the medullary spaces surrounding the teeth (114,115,116,117,118). The periodontal ligament injection is an accepted form of administering local anesthetics which may be used as either a primary or supplemental anesthetic technique (34).

Smith and Walton (114) injected radiocontrast medium and colloidal carbon dye using the periodontal ligament injection in an animal model. The dye was found in the periodontal ligament vessels, medullary bone spaces and the apical foramen of teeth, in the pulps of injected and adjacent teeth, and in the mandibular canal. Smith and Pashley (117) compared the systemic effects, specifically heart rate, blood pressure, and respiration rate, of several different injection techniques and anesthetic solutions in dogs. Epinephrine (1:100,000) alone and 2% lidocaine with 1:100,000 epinephrine produced transient decreases in blood pressure and increases in heart rate whether injected intraosseously, intravascularly, or intraligamentally. These effects were not seen when these solutions were injected submucosally, subcutaneously, intramuscularly, or intrapulpally. Injection of normal saline, 2% lidocaine or 3% mepivacaine caused no systemic effect irrespective of how it was injected.

Kim (119) reported that the periodontal ligament injection causes marked physiologic changes in the pulp including a decrease in blood flow caused by the vasoconstrictor in the anesthetic solution. Perauch (120), Lin et al. (18), and Plamondon et al. (121) found that, following intraosseous injection in animals via the periodontal ligament injection, all pulps were histologically normal. This means that the intraosseous injection is unlikely to cause pulpal necrosis.

The anesthetic efficacy of the intraosseous injection delivered by the periodontal ligament injection has been studied as both a primary and a supplemental injection (19-25). D'Souza et al. (23) studied the intraosseous injection as a primary injection delivered by the periodontal ligament technique. In both maxillary and mandibular second premolars, the teeth mesial and distal to the injected tooth were frequently anesthetized and the anesthesia was more likely to occur with use of a pressure syringe versus a standard syringe.

Schleder et al. (24) used a pressure syringe and the periodontal ligament injection technique in mandibular premolars. They compared 3% mepivacaine, 2% lidocaine, and epinephrine (1:100,00) to 2% lidocaine with 1:100,000 epinephrine. The 2% lidocaine with 1:100,000 epinephrine was significantly superior in gaining anesthetic success as compared to any other solution. The epinephrine (1:100,000) unsuccessfully anesthetized any of the teeth tested. The reported success of the 2% lidocaine with 1:100,000 epinephrine was 87% and the duration of anesthesia was approximately 20 minutes.

Moore et al. (25) used the periodontal ligament injection to compare 2% lidocaine with 1:100,000 epinephrine to sterile normal saline in anesthetizing human mandibular premolars. The sterile normal saline failed to produce any pulpal anesthesia. They reported a 79% success with the anesthetic solution and a duration of anesthesia of approximately 10 minutes.

White et al. (21) studied the intraosseous injection by using the periodontal ligament injection as a primary injection in different maxillary and mandibular teeth. Using a pressure syringe, maxillary and mandibular first molars, first premolars, and lateral incisors were anesthetized with 2% lidocaine with 1:100,000 epinephrine. The success rates of pulpal anesthesia for the maxillary first molars, first premolars, and lateral incisors were 75%, 58.3%, and 39.1%, respectively. The mandibular molars were 79.2%, the premolars 63.2%, and lateral incisors 18.2% successful. The overall duration of anesthesia was found to be ten minutes.

Walton and Abbott (20) used the periodontal ligament injection as a supplement when the primary injection failed to produce adequate anesthesia. They reported a 92% success rate. Smith et al. (22) also used the periodontal ligament technique as a supplementary injection and had a success rate of 83%. In both studies, strong back-pressure was reported as very important in achieving success.

Childers et al. (122) used the periodontal ligament technique to supplement an inferior alveolar nerve block using 2% lidocaine with 1:100,000 epinephrine in both injections. The combination of injections was found to significantly enhance pulpal anesthesia in mandibular first molars for approximately 23 minutes.

Other Intraosseous Techniques

Gaining access to the medullary spaces of the cancellous bone may involve the operator penetrating the mucous membrane, the periosteum, the cortical layer of bone, and finally the medullary bone (123).

Various methods of access to the medullary space have been reported. Archer (124) and Magnes (125) recommended perforation of the cortical bone using an injection needle or the dental handpiece and a small round bur. The use of a handpiece and a root canal reamer was suggested by Lilienthal (126) and Monheim (127). Pearce (128) and Bourke (123) suggested the use of the dental handpiece and a Buetelrock drill to gain access to the desired medullary bone.

Several studies have looked at the use of the intraosseous injection as a primary injection. Lilienthal (126) reported immediate and profound anesthesia in maxillary and mandibular teeth using 2% lidocaine with 1:80,000 noradrenalin. Magnes (125), using 2% lidocaine with 1:100,000 epinephrine, reported clinically effective anesthesia using the intraosseous injection in mandibular anterior teeth. The reported rate of effectiveness was 95-99% in 1,800 patients.

Using the intraosseous injection as a supplemental technique to the inferior alveolar nerve block, Pearce (128) reported 90% effectiveness in mandibular molars undergoing endodontic therapy.

Onset and Duration of Intraosseous Anesthesia

Most clinical studies (123,125,126,128) have confirmed the immediate onset of anesthesia when using the intraosseous injection. Lilienthal (126) reported a 30 to 60 minute anesthetic duration using the intraosseous injection as the primary injection technique. Magnes (125) reported a duration of 1 to 2 hours in all teeth injected. Studies using the electric pulp tester as the means to measure pulpal anesthesia have shown shorter duration times. Moore et al. (25) and White et al. (21) reported approximate durations of 10 minutes. Schleder et al. (24) reported approximately 20 minutes. All of these studies utilized the periodontal ligament injection as the intraosseous technique. Schleder (24) also demonstrated that, without a vasoconstrictor, the duration of anesthesia was one-tenth to one-fifth the time as compared to an anesthetic solution containing a vasoconstrictor.

Soft Tissue Anesthesia

Most authors have reported the lack of lip and tongue anesthesia when the intraosseous injection is given for mandibular anesthesia (123,125,126). However, Matthews et al. (129) reported that 27% of patients receiving the periodontal ligament injection in mandibular teeth did indicate that they had lower lip anesthesia.

STABIDENT INTRAOSSEOUS SYSTEM

The Stabident system is comprised of a handpiece driven perforator which is a solid 27-gauge wire with a beveled end mounted in a plastic, latch-angle shank. This is placed into a latch-angled slow-speed handpiece and activated. The perforator is placed in the desired location of perforation and then activated. A small hole is then made in the cortical layer of bone. A corresponding 27-gauge ultra-short needle is then placed in the perforation and anesthetic solution can then be delivered directly into the cancellous bone surrounding the tooth to be anesthetized (130).

Success

The intraosseous injection using the Stabident system has been studied as both a primary and supplemental injection. Coggins (27) used the Stabident system as a primary injection in different groups of asymptomatic teeth using 2% lidocaine with 1:100,000 epinephrine. He reported an anesthetic success of 93% in maxillary first molars, 90% in maxillary lateral incisors, 75% in mandibular first molars, and 78% in mandibular lateral incisors. Dunbar (26) used the Stabident system as a supplementary injection to an inferior alveolar nerve block in asymptomatic mandibular first molars using 2% lidocaine with 1;100,000 epinephrine. The anesthetic success rate was reported to be 90% for the combination of injections versus 42% for the inferior alveolar nerve block alone. Replogle (28) compared 2% lidocaine with 1:100,000 epinephrine to 3% mepivacaine as a primary injection in

asymptomatic mandibular first molars. The success rate of 3% mepivacaine was only 45% versus 75% using 2% lidocaine with 1:100,000 epinephrine.

Leonard (131) reported a success rate of approximately 89% when using the Stabident system as a primary form of anesthesia in the extraction of teeth. In his study he used 2% lidocaine with 1:100,000 epinephrine and required an average of approximately 1.8 cc of solution per extraction, independent of the arch.

Soft Tissue Anesthesia

In terms of soft tissue numbness, approximately 70% of the patients reported a "numb lip" following a primary injection of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine using the Stabident system (27,28). Coggins (27) and Replogle (28) both reported that subjects felt lip numbness, but it was not as profound as with an inferior alveolar nerve block. They suggested that it is highly doubtful that the inferior alveolar nerve is blocked during the Stabident injection since teeth mesial to the test teeth remained vital to pulp testing. Leonard (131) reported that none of his patients reported lip or tongue numbness after receiving the Stabident injections.

Pain of Infiltration and Intraosseous Injections

Coggins (27) and Replogle(28) reported that the needle insertion for the infiltration injection of the gingiva at the site of perforation caused moderate pain approximately 21% of the time. Deposition of anesthetic solution into the gingiva at the perforation site caused moderate pain in 5% of the patients. They have also reported that, as a primary injection, the perforation of the cortical bone with the Stabident perforator, the insertion of the Stabident needle, and the deposition of

anesthetic solution into the bone resulted in moderate pain in approximately 5% of the patients. Therefore, one can conclude that most patients will report no or mild pain using the Stabident intraosseous technique. As a supplemental injection, after an inferior alveolar nerve block, Dunbar (26) reported very low pain ratings. This is probably due to the anesthetic effect of the nerve block injection on the perforation area.

Onset of Anesthesia

The onset of anesthesia using the Stabident system has been reported to be immediate (26,27,28). There should be no "waiting period" for onset of anesthesia. Dunbar (26) reported that, as a supplemental injection in the first molar, the incidence of slow onset anesthesia (numb after 15 minutes) was reduced from 18% with the inferior alveolar nerve block alone to 0% when the Stabident intraosseous injection was added. Leonard (131) reported anesthesia onset to be 10 to 120 seconds.

Duration of Anesthesia

The duration of anesthesia of the Stabident intraosseous injection, as a primary injection with 1.8 cc of 2% lidocaine with 1:100,000 epinephrine, steadily declined over an hour (27,28). It has been reported that, in 65% of patients, pulpal anesthesia begins to decline at 30 minutes and that at 60 minutes only 40% of patients still have pulpal anesthesia (27). Three percent mepivacaine has an even shorter reported duration of 25% of the patients having pulpal anesthesia at 20 minutes (28). This duration, however, is longer than that reported for the

periodontal ligament injection (21,24,27,28). This is probably due to the increased volume of anesthetic solution being injected into the cancellous bone and/or the increased efficacy of the intraosseous injection. Using the Stabident intraosseous injection as a supplemental injection to the inferior alveolar nerve block, the duration of pulpal anesthesia in mandibular first molars has been reported as very good with 90% of the patients still numb at 60 minutes (27).

Postinjection Pain and Sequela

As a primary and supplemental technique, the majority of patients reported no pain or mild discomfort the day after receiving the intraosseous injection (26,27,28). Approximately 3-10% reported moderate pain (26,27,28). This is less than reported after the use of the periodontal ligament injection (21). It was reported that approximately 4% of patients developed an exudate and/or localized swelling at the site of perforation (26,27,28). Some lasted for weeks, but all resolved with time and/or antibiotic therapy (26,27,28). The slow healing was related to possible overheating of the bone at the perforation site due to the pressure of perforation.

Cardiovascular Effects

Replogle (28) recorded cardiovascular parameters (heart rate and blood pressure) during and 2 minutes after injection of both 3% mepivacaine and 2% lidocaine with 1:100,000 epinephrine using the Stabident system. No significant differences were found in terms of systolic or diastolic blood pressure changes with either solution. Approximately 75% of the patients reported a subjective feeling of

increased heart rate after injection of the 2% lidocaine with 1:100,000 epinephrine solution. No increases in heart rate were reported with the 3% mepivacaine solution (28). Approximately 67% of the patients showed a recorded rise in cardiac rate during or 2 minutes after deposition of the lidocaine solution. The mean heart rate increase was 22 beats/minute from measured rates prior to injection. This usually dissipated within 2 minutes and would be clinically insignificant in healthy patients (28). In 14% of the patients, a mean increase in heart rate of 37 beats from the pre-injection rate was recorded. This result was combined with a prolonged effect of 6 to 22 minutes. These patients may be "epinephrine sensitive," in the sense that the beta effects of the epinephrine on the heart rate caused a greater increase and/or prolonged effect compared to other patients (28).

HUMAN LOCAL ANESTHESIA STUDIES

Local anesthetics have been compared in human experimental studies. These studies have evaluated variations in anesthetic volume, anesthetic concentration, vasoconstrictor concentration, as well as injection techniques.

Mandibular Studies

Wali (132) compared 1.8 ml of 2% lidocaine with 1:50,000 epinephrine, 3.6 ml of 2% lidocaine with 1:50,000 epinephrine, and 1.8 ml of 2% lidocaine with 1:100,000 epinephrine in human inferior alveolar nerve blocks. Vreeland et al. (133) compared the anesthetic efficacy of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine, 3.6 ml of 2% lidocaine with 1:200,000 epinephrine, and 1.8 ml of 4% lidocaine with 1:100,000 epinephrine in human inferior alveolar nerve blocks. Both authors concluded that the success of pulpal anesthesia was not dependent on the volume or concentration of epinephrine and/or lidocaine.

McLean et al. (134) compared equal volumes of 4% prilocaine, 3% mepivacaine, and 2% lidocaine with 1:100,000 epinephrine in human inferior alveolar nerve blocks. Hinkley et al. (135) compared the anesthetic efficacy of equal volumes of 4% prilocaine with 1:200,000 epinephrine, 2% mepivacaine with 1:20,000 levonordefrin, and 2% lidocaine with 1:100,000 epinephrine in human inferior alveolar nerve blocks. No significant differences were found between the anesthetic solutions.

Montagnese et al. (8) compared the Gow-Gates injection to the inferior alveolar nerve block in humans using 1.8 ml of 2 % lidocaine with 1:100,000 epinephrine

and found no difference in the two techniques in gaining anesthesia.

Goldberg (7) compared the inferior alveolar technique to the Akinosi technique and the Gow-Gates technique using 3.6 ml of 2% lidocaine with 1:100,000 epinephrine. He found all three techniques to have similar effectiveness.

Nist et al. (136) compared the inferior alveolar nerve block alone and in combination with the incisive nerve block in human mandibular teeth. The incisive nerve block alone did not anesthetize the central and lateral incisors, but gained anesthesia on the first and second premolars for approximately 30 minutes. The combination of injections produced anesthesia in the premolars for up to one hour and also enhanced pulpal anesthesia in first molars and lateral incisors.

Bilateral inferior alveolar nerve blocks were evaluated in human anterior mandibular teeth by Yonchak (137). His results indicated that cross innervation does exist in the central and lateral mandibular incisors, but that it does not appear to be the primary cause of anesthetic failure in these teeth.

Clark (138) studied the effectiveness of the mylohyoid nerve block alone and in combination with the inferior alveolar nerve block. Using 1.8 ml of 2% lidocaine with 1:100,000 epinephrine, he reported that the mylohyoid nerve block alone was ineffective as a primary injection and did not enhance pulpal anesthesia when combined with the inferior alveolar nerve block.

Simon (139) studied the anesthetic effectiveness of an inferior alveolar nerve block after using a peripheral nerve stimulator to locate the inferior alveolar nerve at the injection site. Hannan (140) used ultrasonic imaging to locate the proper

injection site for an inferior alveolar nerve block. In both studies, the techniques were not any more successful than giving the conventional inferior alveolar nerve block.

Clark (141) compared the anesthetic efficacy of the inferior alveolar nerve block, lingual infiltration, and a combination of the block with labial or lingual infiltrations in human mandibular anterior teeth. The infiltrations alone were not effective in gaining pulpal anesthesia. However, the inferior alveolar block in combination with either a labial or lingual infiltration did increase the success rate of pulpal anesthesia in lateral incisors from 40% to 60%.

Maxillary Studies

Maxillary anesthesia tends to be more clinically successful than mandibular anesthesia. However, problems still exist (1,2,3,34). Anesthetic efficacy has also been extensively studied in the maxillary arch.

Mikesell (142) compared infiltrations of 1.8 ml and 3.6 ml of 2% lidocaine with 1:100,000 epinephrine in maxillary infiltration injections. He found that increasing the volume of anesthetic increased the duration of pulpal anesthesia in maxillary teeth, but that pulpal anesthesia still did not last longer than one hour in these teeth.

Mason (143) compared the anesthetic efficacy of 1.8 ml of 2% lidocaine with either 1:100,000 or 1:50,000 epinephrine and 3% mepivacaine in maxillary infiltration injections. He found that increasing the vasoconstrictor concentration increased duration of pulpal anesthesia in lateral incisors but not first molars. Three percent mepivacaine was found to have a significantly shorter duration than either

of the two lidocaine solutions and none of the solutions could produce anesthesia for over one hour on any of the teeth tested.

Berberich (144) used the infraorbital nerve block in humans to compare the anesthetic effectiveness of 1.8 ml of 2% lidocaine with 1:100,000 or 1:50,000 epinephrine and 3% mepivacaine. He found that the infraorbital block provided anesthesia to the canines, first premolars, and second premolars, but not to the central and lateral incisors or first molars. The study showed no significant differences in the onset of pulpal anesthesia among the solutions tested. Again, the duration of anesthesia was less than one hour.

Knoll-Kohler et al. (145) used 2% lidocaine solutions with either nothing, 1:200,000, 1:100,000, or 1:50,000 epinephrine in maxillary incisors. The lidocaine solutions with 1:100,000 and with 1:200,000 epinephrine decreased the failure rate of anesthesia and increased the duration of effective pulpal anesthesia, but had no effect on the onset time of the anesthetic. No significant differences were observed between 2% lidocaine with 1:100,000 and 1:50,000 epinephrine.

CHAPTER III

METHODS AND MATERIALS

Selected portions of the following methods and materials were derived from procedures developed by previous Examination Committees and presented in Theses by Dreven (29), Uhle (30), and Dunbar (26) in the Department of Endodontics, The Ohio State University.

Fifty-one adult subjects presenting for emergency treatment at The Ohio State University College of Dentistry were used in this study. All subjects were in good health as determined by a written health history and oral questioning. The subjects had no contraindications to the injection techniques, nor the solutions being tested. The study was approved by The Ohio State University Human Subjects Review Committee and written consent was obtained from each subject.

Subjects included in this study had a posterior tooth with a clinical diagnosis of irreversible pulpitis and actively had pain associated with the tooth. The diagnosis was made in the Emergency Clinic at The Ohio State University, College of Dentistry and was reconfirmed by the principal investigator. By definition the teeth were vital and gave a prolonged painful response to thermal testing. Some of the teeth were also sensitive to percussion, had a history of spontaneous pain, and/or had a thickened periodontal ligament

space as determined by a periapical radiograph. The patient was given the option of endodontic treatment or extraction of the tooth, and was given the option of participation or nonparticipation in this study. Teeth which had periapical pathosis, other than a thickened periodontal ligament space, or which were nonvital were not included in this study.

Prior to any testing or treatment, patients were asked to rate the pain of the test tooth on a scale of 0-3. Zero indicated no pain. One indicated mild pain, pain which was recognizable but not discomforting. Two indicated moderate pain, pain which was discomforting but bearable. Three indicated severe pain, pain which caused considerable discomfort and was difficult to bear.

All test teeth and adjacent teeth were tested with the Analytic Technology® Digital Pulp Tester (Analytic Technology, Redmond, WA) and 1,1,1,2 tetrafluoroethane (Green Endo-Ice®, Hygenic Corp., Akron, OH) prior to any anesthesia being given. This served as baseline information.

The Analytic Technology® Digital Electric Pulp Tester had a rate of current increase that was kept constant throughout the study. The elapsed time from zero to the highest reading (80/80) was 25 seconds. Nickel-cadmium batteries (General Electric, Gainesville, FL) were used and recharged after each days use. All testing was performed by the principal investigator.

All pulp testing was performed in the same manner. The principal investigator wore sterile latex examination gloves. The teeth were isolated with cotton rolls and dried with 2x2 cotton gauze. A small amount of Crest Gel toothpaste (Proctor & Gamble Co.,

Cincinnati, OH) was placed on the tip of the electrode to aid in electrical pulse conduction to the teeth. The electrode was placed firmly on sound enamel (in the middle third of the buccal surface, or lingual surface if no intact buccal surface existed) of the clinical crown. The electrode was not placed on restorations, on enamel supported by restorations, or cervically exposed dentin. The testing of each tooth began upon contact of the electrode to the tooth and terminated when the subject raised his or her hand to indicate the initial sensation in the tooth. The value on the digital readout of the pulp tester was then recorded. If the subject felt nothing, even at the maximum value of 80/80, the test was stopped, and a value of 80 was recorded.

Green Endo-Ice ®, 1,1,1,2 tetrafluoroethane (TFE), was also used as a testing agent. The tooth to be tested was again dried with 2x2 cotton gauze. The TFE was sprayed onto a cotton pellet, held by a cotton forceps, until the pellet was saturated. The pellet was then applied to the middle one third of the buccal enamel surface (the lingual surface was used as an alternate site if no buccal surface existed). The pellet was removed as soon as the patient indicated a feeling of cold or pain. A positive response was then recorded. If the patient felt nothing and the TFE had evaporated from the pellet (taking approximately 30 seconds to occur), a "no response" was recorded.

The vitality tests were conducted sequentially, the electric pulp tester then the Green Endo-Ice®, prior to and after anesthesia was given. Beginning with patient 38, patients were asked to rate the pain caused when being tested with the electric pulp tester and the TFE. The responses utilized a pain scale of 0-3. Zero indicated that the subject felt the stimulation but it did not cause any pain. One indicated mild pain in which the subject felt

the stimulation but it only caused a short-term painful sensation. Two represented moderate pain in which the patient responded to the stimulation but it caused an exaggerated and painful, but not prolonged, sensation. Three indicated severe pain in which the subject felt the stimulation and it caused an exaggerated and prolonged (greater than 10 seconds) response.

An injection of one cartridge (1.8 ml) of 2% lidocaine with 1:100,000 epinephrine (Astra Pharmaceutical Products, Inc., Westborough, MA) was given for mandibular teeth and 3.6 ml of the same solution for maxillary teeth by the principal investigator. This consisted of either an inferior alveolar nerve block (IANB) for mandibular teeth, or a buccal infiltration injection for maxillary teeth. Benzocaine topical anesthetic (Hurricaine® gel, Beutlich, Inc., Niles, IL) was applied for approximately 1 minute to the site of injection prior to the administration of anesthetic solution.

The inferior alveolar nerve block was administered using a standard aspirating syringe (Astra Pharmaceutical Products, Inc., Westborough, MA) and a 27-gauge, 1 1/2" needle (Monoject®, Sherwood Medical, St. Louis, MO). The conventional inferior alveolar injection technique as described by Fischer (146) and modified by Jorgensen and Hayden (147) was used. The injection site was the soft tissue overlying the medial surface of the ramus, lateral to the pterygomandibular raphe, at a height determined by the coronoid notch on the anterior border of the ramus. With the subjects mouth wide open, the thumb of the noninjecting hand was placed over the pterygomandibular triangle and then pulled laterally until the deepest depression in the anterior border of the ramus was felt. The first or second finger of the noninjecting hand palpated the posterior portion of the ramus,

finding a slight depression. The line between the thumb and the finger established the vertical height of the injection site. The direction of needle insertion was from the contralateral mandibular premolars and directed parallel to the occlusal plane. The needle was placed just under the mucosal surface (2-3 mm). It was then advanced over a time period of ten seconds to the target site until bone was gently contacted, a depth of penetration of approximately 16 to 20 mm. After contact with bone was made, the needle was withdrawn 1 mm, aspiration performed, and 1.8 ml of the anesthetic solution was deposited over a period of one minute. Positive aspirations were recorded on the questionnaire. Postinjection evaluation timing started after the needle was withdrawn.

The maxillary injection followed a basic infiltration technique as described by Malamed (34). An aspirating syringe and 27-gauge, 1 1/2" needle was used. The orientation of the bevel of the needle was directed toward the bone. The noninjecting hand pulled the lip laterally so that the buccal tissue was taut. The syringe was held parallel with the long axis of the tooth to be anesthetized. The needle was inserted just under the mucosal surface (2-3 mm) in the height of the mucobuccal fold over the target tooth. The needle was advanced until the bevel was at a height of or above the apical region of the tooth. Aspiration was performed and 1.8 ml of anesthetic solution was deposited slowly over one minute. A repeated injection utilizing another 1.8 cc of 2% lidocaine with 1:100,000 epinephrine immediately followed, again depositing the anesthetic solution slowly over a minute. The timing for postinjection evaluation began upon withdrawal of the needle following the second injection.

During all injections the patients were asked to rate the pain they experienced during the injection utilizing a scale of 0-3. This, again, followed the rating scale previously described. Patients indicated the level of pain they experienced by raising their left hand and extending the correlating number of fingers to the scale being used. No pain was indicated by a closed hand. Patients were asked to rate the pain of the injection they received at three distinct periods. One, needle insertion, which was defined as the pain of mucosal perforation. Two, needle placement, which was the pain when the needle was advanced to its specified location. Three, anesthetic deposition, which was the pain during deposition of the anesthetic solution.

Mandibular tooth testing began 1 minute postinjection and was done every minute for 5 minutes utilizing the electric pulp tester and the Green Endo-Ice®. A sequential testing cycle of electric pulp testing followed by Green Endo-Ice® was utilized throughout the testing phase of the study. Beginning with patient 37, patients were asked to rate any pain they felt due to the testing procedures using the same scales as used in the initial testing phase of the study. After 5 minutes, the IANB was evaluated for success in terms of a feeling of lip numbness. Failure to achieve this feeling resulted in a second IANB being given as described and retesting for another 5 minutes. No data was collected for injection pain during the second IANB, but pain ratings from electric pulp testing and testing with Green Endo-Ice® were still conducted. Maxillary teeth were tested in a similar manner and patients rated the test pain (starting with patient 37) every minute, postinjection, for 3 minutes.

If the patient responded to either of the tests, following a "clinically successful" injection, a supplemental intraosseous injection using the Stabident system (Fairfax Dental Inc., Miami, FL) was given distal to the tooth exhibiting the irreversible pulpitis. In second and thrid molars, the intraosseous injection was given mesial to the experimental tooth.

The intraosseous injections were given using the Stabident system and 1.8 ml of 2% lidocaine with 1:100,000 epinephrine. It was recommended in the Stabident Instruction Manual (130) that the injection be given distal to the tooth intended to be anesthetized and this was followed except for second and third molars. The area of perforation of the cortical plate of bone was determined by the horizontal line of the buccal gingival margins of the experimental and adjacent teeth and a vertical line that passed through the interdental papilla. A point approximately 2 mm below the intersection of these lines, in the attached gingiva, was used as the perforation site.

An infiltration injection of approximately 0.1 ml of 2% lidocaine with 1:100,000 epinephrine was deposited using a 27-gauge ultra-short Stabident needle at the site of perforation. This led to a blanching of the tissue. The cortical bone was then perforated approximately 1 minute later using the Stabident perforator in a contra-angle, slow-speed handpiece. The perforator was inserted into the hole created by the 27-gauge needle and was perpendicular to the cortical plate. Using light pressure, the handpiece was activated until a "break through" feeling was observed, or until 2-5 seconds had elapsed. If this feeling was not achieved, the angle and position of the perforator was altered and penetration reattempted.

The perforator was removed while still activated and a cotton roll was applied to the area to help reveal the location of the perforation and control hemorrhage. A 27-gauge ultra-short Stabident needle, attached to an aspirating syringe, was then inserted into the perforation and 1.8 ml of 2% lidocaine with 1:100,000 epinephrine was delivered over 2 minutes. If back-pressure occurred during delivery of the anesthetic solution, it was recorded. Patients were advised prior to receiving the injection that they may feel an increase in their heart rate after receiving the intraosseous injection and that it would subside after approximately 2 minutes. During and for 2 minutes after the intraosseous injection, the patient was questioned and any positive response to this subjective increase in heart rate was recorded.

Patients were again asked to rate the pain they experienced during the intraosseous injecting using the pain scale previously described. The patients indicated their subjective level of pain utilizing the previously described hand signals. Patients were asked to rate the discomfort, if any, during the insertion of the needle for the gingival infiltration; the deposition of the anesthetic solution in the gingiva; the perforation of the cortical bone; and the deposition of the anesthetic into the cancellous bone.

Upon completion of the intraosseous injection, the tooth was tested every minute with both the electric pulp tester and Green Endo-Ice®, alternately, for 3 minutes. Again, starting with patient 37, subjects were asked to rate any pain they experienced during the electrical and thermal tests.

If a positive response to either the electric pulp tester or the Green Endo-Ice® was recorded at the end of the three minute testing cycle, following the initial intraosseous

injection, additional supplemental injections (repeated distal intraosseous injection; mesial intraosseous injection; or intrapulpal injection) were performed. If a second intraosseous injection was required, patients were again asked to rate any pain they experienced during the intraosseous injection (as previously described) and during thermal and electric pulp testing. Testing was repeated, as above, for another three minutes.

Following negative responses to both tests, no response to TFE application and an 80/80 reading with the electric pulp tester at the end of the testing cycle, the tooth was isolated with a rubber dam and the pulp accessed through the occlusal surface of the tooth with a number 4 round bur using a high-speed handpiece. If the patient felt any discomfort during the procedure, they were asked to rate the pain they felt using the aforementioned pain scale of 0-3. Recordings were also made as to the level of access achieved when the patient felt pain. These were recorded as either entering dentin, entering the pulp chamber, removing the pulp (placing files to length), or instrumenting the canals. If the patient had received an intraosseous injection, the time the pain occurred following the completion of the Stabident injection was also recorded.

If the patient reported any pain during the endodontic procedure, supplemental anesthesia was given. The form of this anesthesia depended on what type of injections the patient had received and the stage of the access or instrumentation procedure. If no supplemental injections had been given, and the operator had not yet accessed the pulp chamber (pulp exposure), an intraosseous injection was given. If the pulp chamber had already been opened, an intrapulpal injection was given. The patients were asked to rate the pain of all intrapulpal injections that were required.

If the patient had already received an intraosseous injection, but felt pain prior to pulpal access, a second intraosseous injection was given at a site mesial to the experimental tooth. If the pulp was exposed, an intrapulpal injection was given. Finally, if the patient had received two intraosseous injections and still felt pain during pulpal access, the patient received an intrapulpal injection.

The intrapulpal injection consisted of inserting a 27-gauge, 1" or 1 1/2" needle into the exposed pulp chamber or root canals and wedging it into the chamber or canal. The anesthetic solution was then slowly injected under strong back-pressure for approximately 30 seconds. The patient was warned prior to receiving the injection that there may be a sharp, stabbing pain upon insertion of the needle, but that it would subside within 3-5 seconds after the injection had been started.

The success of the intraosseous injection was defined as endodontic treatment that could be successfully completed without patient discomfort. The success and failure of the primary injections (IANB and infiltrations) were defined by the need for any supplemental injections including the intraosseous or intrapulpal injections. No teeth that were found to be necrotic upon access were included in the study results.

The emergency treatment fee of $50.00 was refunded to the patient for participating in the study.

The data from this study was collected and statistically analyzed. Differences in the arches in terms of need for the intraosseous injection was assessed by the chi-square test. The correlation of vitality test results of the electric pulp tester versus the Green Endo-Ice® was done using a stratified analysis to determine the phi coefficient. Also, a

percentage of agreement was determined between the two tests and a kappa statistic calculated to determine the level of agreement beyond chnace. The success of the Stabident intraosseous injection was assessed by the Fisher Exact test and confidence intervals for success were also calculated.

CHAPTER IV

RESULTS

The raw data was compiled in Appendices A through C. A summary of the results is presented in Tables 1 to 34. The data recording sheet, the consent form, and the Human Subjects Review Committee form are found in Appendices D, E, and F, respectively.

Biographical raw data can be found in Appendix A. A summary of the biographical information can be found in Table 1. Data was analyzed for a total of fifty-one subjects. The mean age was 34.0 years with a range of 19-68 years. Twenty-three subjects (45%) were male and twenty-eight subjects (55%) were female.

The subjects requiring an intraosseous injection consisted of 10 males (42%) and 14 females (58%). The mean age of these 24 subjects was 32.6 years with a range of 19-63 years.

Raw data on tooth type can be found in Appendix A and a summary can be found in Table 2. Twenty-five teeth used in this study were maxillary teeth and the remaining 26 were mandibular teeth. The mandibular teeth consisted of 2 premolars (one first and one second), twelve first molars, eleven second molars, and one third molar. The maxillary teeth consisted of eleven premolars (one first and ten second), twelve first molars, and two

second molars.

Clinical Diagnostic raw data is found in Appendix A. A summary of Clinical Diagnostic raw data is found in Table 3.

All the subjects had pain associated with the test tooth. Eight subjects rated their pain as mild, 19 as moderate, and 24 as severe. The mean pain rating was 2.3 while the median was 2.

All of the experimental teeth were vital as determined by electric pulp tests. The mean baseline EPT reading was 39.5. Starting with patient number 38, patients rated the pain caused by the electric pulp tester. Ten patients rated the pain as mild, two as moderate, and two as severe. The mean pain rating caused by the electric pulp tester was 1.4 and the median was 1.

Forty-nine of 51 subjects responded to thermal testing with Green Endo-Ice®. Eleven subjects (out of 14) rated their pain with Green Endo-Ice® as severe, two rated the pain as moderate, and one subject had no pain with Green Endo-Ice®. The mean pain rating caused by the thermal testing was 2.6 and the median was 3.

Comparing the pain ratings caused by the electric pulp tester versus the Green Endo-Ice®, there was a significant difference found between the two ($p = 0.0039$). The Green Endo-Ice® caused significantly more pain on initial testing. The median difference was -2 with a Test Statistic of -31.

Raw data, utilizing a numerical scale, for all initial injection discomfort ratings is located in Appendix B. Table 4 provides a summary of the pain ratings for needle insertion during the inferior alveolar nerve block (IANB) and infiltration injections for all

experimental teeth. Five patients experienced no pain, 15 experienced mild discomfort, and 6 experienced moderate pain on receiving the inferior alveolar nerve block. For maxillary teeth, 11 subjects experienced no pain, 12 subjects reported mild pain, and 2 subjects experienced moderate pain. No subject rated the pain experienced during initial needle insertion in either injection type as severe.

The summary for pain ratings for needle placement during either the inferior alveolar nerve block or the maxillary infiltration is found in Table 5. For maxillary teeth, 15 patients reported no pain and 10 patients reported mild pain. No patient reported moderate or severe pain on needle placement during the infiltration. For mandibular teeth, 8 patients reported no pain on needle placement, 9 reported mild pain, 5 reported moderate pain, and 4 reported severe pain.

Pain ratings for deposition of the anesthetic solution during the inferior alveolar nerve block and maxillary infiltration are summarized in Table 6. No pain was experienced by 11 patients receiving the IANB, 7 subjects reported mild pain, 6 reported moderate pain, and 2 reported severe pain. For maxillary infiltrations, 17 subjects reported no pain, 6 reported mild pain and 2 reported moderate pain. No subject reported severe pain on deposition of anesthetic solution during the maxillary infiltration.

Raw data from the electric pulp test (EPT) readings and Green Endo-Ice® responses of the experimental teeth is found in Appendix B. The numbers and percentages of experimental teeth with readings of 80/80 and negative responses to Green Endo-Ice® at each postinjection time are summarized in Table 7. For mandibular teeth, at the end of testing (minute 5) 12 teeth (46%) tested 80/80 and 21 teeth (81%) tested negative to

Green Endo-Ice®. Seven patients required a second IANB due to an unsuccessful initial injection. Initial data on these patients was excluded from Table 7. The test data accumulated during testing after the second IANB injection was included in Table 7. For maxillary teeth, at the end of 3 minutes, 23 teeth (92%) tested 80/80 with the EPT and 23 teeth (92%) tested negative to Green Endo-Ice®. Combining these end-of-testing results gave 35 teeth (67%) testing 80/80 and 44 teeth (86%) responding negatively to Green Endo-Ice®. Table 8 summarizes the comparison of the electric pulp tester to Green Endo-Ice®. The level of agreement between the two tests, as determined by the phi coefficient, was 0.648. This indicates a moderate level of agreement between the two testing methods. The percentage of agreement was found to be 80.39% and the kappa statistic was 0.617. Overall for maxillary and mandibular teeth, this shows a moderate level of agreement between the two tests in determining pulpal anesthesia.

Raw data associated with the pain ratings during testing with the EPT and Green Endo-Ice® after the inferior alveolar nerve block or infiltration injections is found in Appendix B. A summary of these pain ratings is found in Table 9. Subjects reported either no pain or mild pain when being tested with the EPT. Only one subject initially rated the EPT testing as moderately painful and that was at the first test interval. Utilizing the Green Endo-Ice® resulted in 2 initially severe responses and 1 severe response at minute 2. After that time interval, subjects rated the pain caused by the Green Endo-Ice® as either mild or no pain.

Raw data for anesthetic failures of the inferior alveolar nerve block and maxillary infiltration is located in Appendix B. A summary of this data is found in Tables 10

through 14. Eighteen of 51 teeth (35%) required a supplemental injection due to a positive reading of the electric pulp tester and/or the Green Endo-Ice® after the final test period (Table 10). Two of these teeth were maxillary and 16 were mandibular. Table 11 summarizes how many teeth required supplemental anesthesia after recording an 80/80 with the electric pulp tester and testing negative to Green Endo-Ice®. Five of 33 (15%) teeth needed further anesthesia due the patients experiencing pain while accessing through dentin (Table 12). Three of 33 (9%) patients had pain when the pulp was entered, 5 (15%) when the pulp was removed, and 1 (3%) during filing (Table 13). Eight of these 14 subjects received intraosseous injections (Table 13) while the remaining 6 received intrapulpal injections (Tables 12 and 13). A total of twenty-four subjects received an intraosseous injection and 8 received an intrapulpal injection (Table 14). Three maxillary teeth and 21 mandibular teeth required an intraosseous injection (Table 14).

Table 15 summarizes the distribution of teeth requiring an intraosseous injection. This group of teeth consisted of 21 mandibular teeth and 3 maxillary teeth. Statistical analysis of this distribution revealed that there was a significant difference ($p = 0.0000$) between the two arches in terms of requiring an intraosseous, supplemental injection. The mandibular teeth consisted of one first premolar, nine first molars, ten second molars, and one third molar. The maxillary teeth consisted of one second premolar, one first molar, and one second molar.

Raw data relative to the pain ratings reported by patients who had pain during initial access or endodontic treatment is located in Appendix B. A summary of the pain ratings and at what stage of treatment they occurred is given in Table 16. Of the 5 patients

reporting pain during access of dentin, 4 reported moderate pain and one reported mild pain. No one reported severe pain. Of the 4 patients experiencing pain upon entering the pulp, 1 reported mild pain, 1 reported moderate pain, and 1 reported severe pain. Upon removing the pulp, 1 patient reported mild pain and 3 reported moderate pain, and 1 reported severe pain. When filing the experimental tooth, one patient reported mild pain.

Eight patients received intrapulpal injections after inferior alveolar or infiltration injections. The raw data of the pain ratings is located in Appendix B. A summary of the pain ratings is given in Table 17. One patient reported no pain, 4 reported mild pain and 3 reported moderate pain on receiving an intrapulpal injection. No patient reported the pain as severe.

Raw data relating to the Stabident intraosseous injection is located in Appendix C. Table 18 provides a summary of the pain ratings for needle insertion during the infiltration injection prior to the intraosseous injection. Ten patients (42%) of 24 experienced no pain, 11 (46%) experienced mild pain, 2 (8%) reported moderate pain, and 1 (4%) reported severe pain.

The summary of pain ratings for anesthetic solution deposition during the aforementioned infiltration is provided in Table 19. Twenty-two (92%) of the patients reported no pain on solution deposition. Two patients (8%) reported a painful experience with 1 being moderate and the other severe.

Pain ratings for the perforation of the cortical plate are summarized in Table 20. No pain was reported by 17 (71%) of the patients. Five patients (21%) reported mild pain, 1 (4%) reported moderate pain, and 1 (4%) reported severe pain.

The summary of pain ratings for anesthetic solution deposition during the intraosseous injection is reported in Table 21. Eighteen (75%) of the patients experienced no pain, 5 (21%) experienced mild pain, and 1 (4%) reported severe pain. No subject reported moderate pain during solution deposition.

Table 22 summarizes the number of subjects in which back-pressure was experienced during the delivery of anesthetic solution during the intraosseous injection. Three (12.5%) of the injections had back-pressure.

Table 23 summarizes the number of patients reporting a subjective increase in heart rate during or immediately after receiving the intraosseous injection. A total of 11 of 24 (46%) patients reported an increase in heart rate upon receiving the intraosseous injection.

Raw data from the electric pulp test readings and Green Endo-Ice® responses is found in Appendix C. Table 24 gives a summary of the numbers and percentages of experimental teeth that had readings of 80/80 and a negative response to Green Endo-Ice® at each post-intraosseous injection time. At the end of the testing period (3 minutes), 23 patients (96%) tested 80/80 with EPT and 24 patients (100%) were negative to Green Endo-Ice®.

Raw data for pulp testing and clinical results after the Stabident intraosseous injection is found in Appendix C. Tables 25 through 29 summarize the pulp testing and clinical results following the initial intraosseous injection. Table 25 shows that 1 of 24 (4%) patients tested positive to the electric pulp tester after receiving the intraosseous injection. Table 26 shows that 6 of 24 (25%) patients required a second supplemental injection due to pain during endodontic therapy. This includes the patient from Table 25 and patients

who were considered a success for the intraosseous injection. Table 27 shows that 1 of 23 (4%) patients reported pain on accessing the dentin. Table 28 shows that 4 of 23 (17%) patients reported pain upon entering the pulp, and 1 of 23 (4%) reported pain upon removal of the pulp. No patient reported pain while filing the canals. Of the 6 patients requiring a second supplemental injection, 2 of 24 (8%) received second intraosseous injections and 4 of 24 (17%) received intrapulpal injections (Table 29). One of the two patients requiring a second intraosseous injection was the patient who responded positively to the electric pulp tester (Table 25). This patient had endodontic therapy started, even though she did not gain an 80/80 reading with the electric pulp tester. This patient had pain upon entering the pulp.

Table 30 summarizes the pain ratings reported during access or endodontic therapy following the initial intraosseous injection. One patient reported moderate pain when the dentin was accessed, two reported mild pain and one moderate pain upon the pulp being exposed, and two reported moderate pain when the pulp was removed.

Table 31 summarizes the pulp testing results following the administration of a second Stabident intraosseous injection. One patient (the same patient as in Table 25) tested positive with the electric pulp tester but negative to Green Endo-Ice®. This patient received treatment despite this result.

Table 32 summarizes the clinical results following a second intraosseous injection. The one patient who tested negative to both the electric pulp tester and Green Endo-Ice®, following the second intraosseous injection, had no pain during root canal treatment. The

patient who tested positive with the electric pulp tester reported pain when the pulp was entered and required an intrapulpal injection.

A summary of the time, post-intraosseous injection, when the patients experienced pain during access or endodontic therapy is given in Table 33. Table 33 also provides a summary of the secondary supplemental anesthesia given and the pain ratings reported if and when the patient received an intrapulpal injection. Patients numbers 4 and 25 were considered successes in terms of intraosseous anesthesia due to length of time, post-intraosseous injection, when they felt pain and because they rated the pain of the secondary intrapulpal injection as none. The mean time of the remaining four subjects when pain was experienced was 5:04. Two of these subjects received secondary intrapulpal injections and two received secondary intraosseous injections. The two patients who received intrapulpal injections both rated them as mild in terms of pain experienced.

The raw data for the two patients receiving secondary intraosseous injections is found in Appendix C. One patient reported moderate pain during a second perforation while the remaining injection procedures were rated as nonpainful. One subject experienced subjective heart rate increase. In both cases, no back-pressure was experienced during injection of the anesthetic solution into the second perforation. Subject #8 responded to electric pulp testing during all three postinjection time intervals while subject #5 tested 80/80 for all three intervals. Neither patient responded to testing with Green Endo-Ice® during the entire secondary test period. Patient #5 did not require any subsequent supplemental anesthesia (Table 33). Patient #8 experienced moderate pain, 3:25 after the

second intraosseous injection, upon entering the pulp and received an intrapulpal injection which was rated as moderately painful (Tables 33).

Table 34 summarizes the success and failure of the Stabident intraosseous injection. In maxillary teeth, the injection was 66.67% successful. In mandibular teeth the intraosseous injection was 90.48% successful. This results in a combined success rate of 87.5%. The Fisher Exact Test was used to assess differences in the distribution of success and was not significant ($p = 0.343$). In terms of relating our results to the general population, for a 95% confidence interval for mandibular success, the population range can run from 69.81% to 98.82%. For a 99% confidence interval for mandibular success, the range of population success is 63.14% to 99.50%. Calculations for maxillary confidence intervals could not be done due to the sample size being too small.

CHAPTER V

DISCUSSION

Selected portions of the following Discussion section were derived from previous Theses by Uhle (30), Dunbar (26), Coggins (27), Replogle (28), and Dreven (29) in the Department of Endodontics, The Ohio State University.

Methods and Materials

Fifty-one subjects participated in this study of which 23 were male and 28 were female. The population of subjects came from patients seen in The Ohio State University, College of Dentistry Emergency clinic and gives a relatively good representation of the general population in terms of those patients whom experience difficulty obtaining profound anesthesia. The age range was from 19 to 68 with the mean age being 34.0 years. No attempts were made to balance the number of male versus female subjects, nor the ages of the patients. These factors have been shown to have no effect on electric pulp testing thresholds (10,148) and the effects these factors would have on obtaining complete anesthesia is not known.

A clinical setting using a human test model was used because animal studies cannot be directly correlated for anesthetic effect in humans. For this reason, the human test model was used to allow direct correlation between anesthetic techniques.

All subjects were compensated for their participation by having their emergency endodontic fee ($50.00) refunded to them. All subjects were determined to be in good health by a written health history and oral questioning (Appendix G). Participation in this study was voluntary in accordance with The Ohio State University Human Subjects Committee. All patients received prompt emergency treatment whether or not they participated in this study. Subject motivation may have been altruistic or due to the compensation they received.

Posterior teeth, both maxillary and mandibular molars and premolars, were used exclusively in this study. No attempt was made to balance the number or type of posterior teeth used. Although differences in pain thresholds are present between molars and premolars (10,148), the effects these differences have on teeth in obtaining complete pulpal anesthesia is not known. White et al. (21) reported on the efficacy of the intraosseous injection, by way of the periodontal ligament injection, and found that success of the injection decreased from posterior to anterior teeth from a high of 79.2% in mandibular molars to a low of 18.2% in mandibular lateral incisors. Their study utilized normal maxillary and mandibular teeth and the Analytic Technology® pulp tester. They postulated that as the size and number of cribiform plate perforations decreased from the posterior region to the anterior region (149), so did the effectiveness of the injection. No overall differences were found between the success rates of maxillary (56.7%) versus mandibular (56%) teeth. Coggins (27) reported that there was no significant differences in the incidence of pulpal anesthesia, duration of anesthesia, nor relative onset of anesthesia utilizing the Stabident intraosseous technique between mandibular and maxillary first

molars. However, he reported that there was a significant difference in the anesthetic success between mandibular (75%) and maxillary (93%) first molars (27).

All teeth used in this study were diagnosed with clinical irreversible pulpitis including spontaneous pain which was rated as mild to severe. Wallace et al. (150) surveyed diplomates of the American Association of Endodontists and found that 235 of 268 reported that patients with an acutely painful tooth still had pain on opening the tooth even when the patient had signs of lip and tongue numbness. They also reported that 210 of the 268 diplomates felt that regional anesthesia was more difficult for a painful tooth than a nonpainful tooth.

Seltzer et al. (88), Mendoza et al. (112,113), and Mitchell and Tarplee (109) have reported that a clinical diagnosis cannot be precisely correlated to a pulpal, histological condition. However, Seltzer (111) stated that severe and spontaneous pain appears to indicate the presence of severe pulpal pathosis. Lundy and Stanley (110) also found severe clinical responses to accompany acute states of pulpal histology. Mendoza et al. (112,113) demonstrated that teeth diagnosed with irreversible pulpitis had moderate to severe changes in nervous, vascular, and connective tissues of the apical pulp tissue. It would appear that these pulps were inflamed. Cooper (151) has reported that if pain is intermittent, pain relief may be wrongfully attributed to the drug being tested. Perhaps this would also be true if anesthesia was being measured clinically. Therefore, all patients tested had a history of constant, spontaneous pain.

Prior to any pulp testing, patients were asked to rate the pain they were experiencing with the test tooth on a scale of 0 to 3. Zero indicated no pain. One indicated mild pain,

pain which was recognizable but not discomforting. Two indicated moderate pain, pain which was discomforting but bearable. Three indicated severe pain, pain which caused considerable discomfort and was difficult to bear. This was done to ensure that patients met the criteria of having spontaneous pain immediately prior to the beginning of the study.

The Analytic Technology® electric pulp tester was used as one method to evaluate pulpal anesthesia in this study. Bjorn (2) recommended the use of the electric pulp tester to evaluate the effects of dental local anesthesia. Cooley et al. (72) and Kitamura et al. (71) have shown that this device is highly accurate in predicting the vitality of teeth. McDaniel et al. (12) showed that the use of the electric pulp tester does not cause structural changes nor pathosis in pulpal tissue. Dreven et al. (11,29) reported that a reading of 80/80 correlated with clinical analgesia in normal and asymptomatic teeth. The electric pulp tester was found to be capable of accurately evaluating local anesthetic techniques and solutions experimentally in normal teeth. Dreven et al. (11,29) also found that 27% of teeth with irreversible pulpitis did not have profound anesthesia even after an 80/80 reading. Increases in electric pulp tester readings above baseline readings, but without attaining 80/80, may not clinically indicate profound pulpal anesthesia.

The electric pulp tester has an internal resistance of 150 Kohms which negates the high resistance in teeth as reported by Bjorn (10), Cooley and Robison (67), Mumford and Newton (68), and Mumford and Bjorn (152). Matthews et al. (65,153) showed that 140 volts was sufficient stimuli for all vital pulps in normal, asymptomatic teeth. The Analytic Technology® electric pulp tester produces 300 volts which more than meets this

requirement. The Analytic Technology® pulp tester produces a maximum output of 50 microamps. Pepper and Smith (154) and Matthews et al. (155) found 50 microamps sufficient to stimulate healthy pulps and that 200 microamps was required to stimulate the surrounding periodontal tissues. Matthews et al. (155) also recommended the use of constant current stimulation rather than impulse current stimulation. This criteria, which the Analytic Technology® electric pulp tester provides, allows for current to remain stable even if variable resistance's are encountered in tooth structure (64).

The rate of voltage increase during this study was calibrated on the electric pulp tester so that the elapsed time to go from a 0 reading to a maximum reading of 80 was approximately 25 seconds. This rate of increase was selected based on work by Kleier et al. (156) who reported that a slow rise in voltage output (25 seconds) resulted in significantly less painful responses versus a more rapid rise (5 seconds). Nickel-cadmium batteries were used and recharged after each day's use to ensure adequate power to the pulp tester for the duration of testing.

All pulp testing was done by the primary investigator in the same manner. The tester wore latex examination gloves to comply with infection control guidelines. The teeth to be tested were isolated with cotton rolls and dried with 2x2 cotton gauze. This was done to prevent shunting of electrical stimulus which could lead to negative responses as shown by Cooley et al. (72) and Cooley and Robison (67). Stephan (157) and Nahri et al. (158) reported that if the test teeth were not adequately dried, the periodontal tissues could be stimulated and produce a false positive response. The electrode tip was coated with a small amount of Crest gel toothpaste to aid in conduction of the electrical pulse to the

teeth. Martin et al. (159) found no difference among different brands of toothpaste used as a conduction medium for the electric pulp tester. Crest gel was chosen because of its higher viscosity which minimized flow, its tendency not to "cake up" on the teeth and electrode tip, and its water solubility which allowed for easy removal from the teeth and electrode with a wet gauze.

The electrode tip was placed firmly on sound enamel of the middle third of the buccal surface of the tooth. If caries or restorations prevented this, the electrode was placed on the lingual surface or any intact enamel present. This position was then used throughout testing on that particular subject. The effect of caries on the electric pulp tester readings is not known. Cooley and Robison (67) reported that the electrode should be placed flat against the enamel versus being rotated. This would allow maximum voltage transmission to the tooth to be obtained. Jacobson (160) evaluated probe placement sites on extracted teeth using an Analytic Technology® pulp tester and an oscilloscope. He found that the best location for the probe was the occlusal two-thirds of the tooth. More specifically, the middle third of incisors and the occlusal third of premolars were shown to have the least resistance and placement here reduced the possibility of false positive readings from gingival nerve fiber stimulation. The electrode, in this study, was not placed on restorations, enamel supported by restorations, or cervically exposed dentin.

The Analytic Technology® electric pulp tester is supplied with an electrical lead that is attached to the electrode and to a lip clip that is placed on the patient's lip or held in their hand. This acts to complete the circuit of the electric pulp tester. Cailleteau et al. (161) and Anderson et al. (162) found that pulp test readings were accurate when patients held

the electrode when it was in contact with a tooth. Since the electrical lead is attached to the probe, and the only requirement is completion of a circuit, the patients were allowed to hold the lip clip with their fingers. The testing of each tooth was started upon contact of the electrode to the tooth and terminated when the subject raised his/her hand to indicate the initial sensation in the tooth. The value on the digital readout was then recorded. If the subject felt nothing, even at the maximum value of 80/80, the test was stopped and a value of 80 was recorded.

The experimental tooth and adjacent teeth were also tested initially with Green Endo-Ice®. This product consists of 1,1,1,2 tetrafluoroethane and a fragrance. This was done to confirm the clinical diagnosis of irreversible pulpitis (13,14,15) and pulpal anesthesia (17). Green Endo-Ice® was selected due to its reported low liquid temperature (-26.2°C) (89) and the fact that skin refrigerants have been found to be more reliable than ice or ethyl chloride in determining pulpal vitality (16). Previous studies (16,17,163) utilized dichlorodifluoromethane as a testing agent. However, this product is no longer commercially available due to its potential environmental hazard and has been replaced by 1,1,1,2 tetrafluoroethane. This material has not been reported on in any studies.

After electric pulp testing, the test tooth was dried again prior to thermal testing. A cotton pellet was saturated with the Green Endo-Ice® and placed in the same location on the tooth as the electrode for the electric pulp tester. Care was taken not to get any of the material on the gingiva. The patient was asked to raise his/her hand when a cold or painful sensation was felt. The pellet was then immediately removed and a positive response was recorded. If no response occurred, the pellet was left on the tooth until the

refrigerant evaporated and a "no response" was recorded. This took approximately 30 seconds. Beginning with patient 38, patients were asked to rate any pain in their responses to both the electrical and thermal tests during initial pulp testing. The scale used was as follows: (0) none - the subject felt the stimulation but it did not cause any pain; (1) mild - the subject felt the stimulation but it only caused a short-term painful sensation; (2) moderate - the subject's response to the stimulation was exaggerated and painful, but not prolonged; (3) severe - the subject's response to either stimulation was both exaggerated and prolonged (greater than 10 seconds). Seltzer et al. (164) have shown that teeth that have a painful and prolonged reaction to thermal stimulation require either root canal therapy or extraction.

Hurricaine® gel topical anesthetic was used prior to the inferior alveolar nerve block and maxillary infiltration injections. This was done to show the patients that everything was being done to prevent discomfort. Yonchak (137), Nist et al. (136), Mikesell (142), and Mason (143) reported that the application of topical anesthetic had no significant effect on pain ratings of injections as compared to placebo. However, their patients were not in pain or apprehensive about treatment they were about to receive.

The inferior alveolar nerve block utilized in this study was given in the manner described by Fischer (146) and modified by Jorgensen and Hayden (147). The description of the inferior alveolar nerve block technique was repeated from the Materials and Methods of Wali (132), Hinkley et al. (135), McLean et al. (134), Vreeland et al. (133), Clark (138), Nist et al. (136), Yonchak (137), Goldberg (7), and Childers (122). All of

these studies utilized the Analytic Technology® pulp tester to determine the effectiveness of the inferior alveolar nerve block in normal teeth.

Subjects with mandibular teeth diagnosed with irreversible pulpitis were placed in the supine position with the neck extended and the mouth opened as wide as possible when the inferior alveolar nerve block was given. The injection site was the soft tissue overlying the medial surface of the ramus, lateral to the pterygomandibular raphe, at a height determined by the coronal notch on the anterior border of the ramus (133). After applying Hurricaine® topical anesthetic to the area for 1 minute, a 27-gauge 1 1/2" needle was inserted 2-3 mm below the mucosal surface coming from the contralateral mandibular premolar area and being directed parallel to the occlusal plane. The needle was advanced over a time period of 10 seconds until bone was gently contacted and then withdrawn 1 mm and aspiration performed. Each patient received 1.8 cc of 2% lidocaine with 1:100,000 epinephrine deposited over 1 minute. This volume and concentration was chosen because Wali (132) and Vreeland et al. (133) found that increasing the volume or the concentration either of the epinephrine or lidocaine above 1.8 cc of 2% lidocaine with 1:100,000 epinephrine did not statistically increase the incidence or success of pulpal anesthesia. Thus, a minimum dose and concentration was used in this study as well as a brand and form that is readily available and often used clinically.

Lip and tongue anesthesia was evaluated subjectively by asking the patient if his/her lip and tongue were numb after each testing cycle. These questions, especially the lip numbness, helped evaluate whether the inferior alveolar nerve block could be judged to be clinically successful. Soft tissue testing, in the form of mucosal sticks, were not performed

in this study. Testing in this manner has not been proven to be accurate in determining anesthesia (165). Wali (132), Goldberg (7), Vreeland et al. (133), Hinkley et al. (135), and McLean et al. (134) compared the incidence of soft tissue anesthesia, mucosal sticks, and pulpal anesthesia and found them to show different rates of success. These authors concluded that mucosal sticks were of little value in determining pulpal anesthesia. In this study, the subjective signs of numbness were evaluated each minute for 5 minutes post-inferior alveolar nerve block injection. If subjective signs were not achieved, a second alveolar nerve block was given using 1.8 cc of 2% lidocaine with 1:100,000 epinephrine and a second 5 minute set of testing cycles was begun. Seven of 25 mandibular subjects (28%) required a second injection. None of these patients required a third injection. The use of a five minute waiting time was based on studies by Goldberg (7) and Nist et al. (136) who reported the average onset times of subjective lip numbness as 5.3 and 5.6 minutes, respectively.

The maxillary injections followed a basic infiltration technique as described by Malamed (34). An aspirating syringe with a 27-gauge 1 1/2" needle was used to inject 3.6 cc of 2% lidocaine with 1:100,000 epinephrine. The bevel of the needle was oriented toward the bone and the syringe was held parallel to the long axis of the tooth to be anesthetized. The needle was inserted 2-3 mm under the mucosa, again after a 1 minute application of Hurricaine® topical anesthetic, in the height of the mucobuccal fold and advanced to a height of or above the apical region of the tooth. The area was aspirated and the anesthetic solution was deposited over 1 minute to reduce patient discomfort. Mikesell (142) found that 3.6 cc of 2% lidocaine with 1:100,000 epinephrine increased the

success rate of pulpal anesthesia to 84% as compared to 1.8 cc of 2% lidocaine with 1:100,000 epinephrine which was successful at a rate of only 38% in maxillary first molars. Mason (143) reported that increasing the concentration of epinephrine to 1:50,000 did not increase the anesthetic success rate in maxillary molars. Vitality and thermal testing was done, as described for mandibular teeth, every minute for 3 minutes. This was deemed to be a clinically reasonable time to wait for maxillary anesthesia to take effect.

While subjects received either the inferior alveolar nerve block or the maxillary infiltration, they were asked to rate the discomfort they experienced at three distinct stages of the injection. This included the time when the needle was placed submucosally, the placement of the needle to its final location, and the deposition of the anesthetic solution. The subjects were asked to hold up fingers indicating discomfort on a zero to three pain scale. A closed hand was used to indicate no pain. One related to mild pain, (recognizable but not discomforting). Two corresponded to moderate pain, (discomforting but bearable). Three related to severe pain, (considerable discomfort and difficult to bear). This rating scale maintained a continuity with previous studies (7,8,11,21,24,25,26,27,28,122,132-138,141,142,143,144). A numerical rating scale was used since Goldberg (7) and Nist et al. (136) showed that there were no differences between it and a visual analog scale.

Electric pulp testing and testing with Green Endo-Ice® was done every minute for 5 minutes on mandibular teeth and 3 minutes for maxillary teeth in a sequential cycle. A stopwatch was started upon withdrawal of the needle after the last injection. It took

approximately 30 seconds to complete the electric pulp test if a reading of 80/80 was achieved and approximately 30 seconds for the Green Endo-Ice® to evaporate from the cotton pledget in the patient's mouth if no response was achieved. Pantera et al. (163) found that the use of dichlorodifluoromethane (-50°C) did not affect subsequent electric pulp test readings or cause false negative responses. Beginning with patient 37, patients were asked to rate any pain they experienced due to either the electric pulp tester or the Green Endo-Ice®. The scale used was the same as previously described for pain ratings of initial Green Endo-Ice® and vitality tests.

After the testing phase was completed, a determination of anesthesia was made. If, for mandibular teeth, no lip and tongue anesthesia was achieved, a second inferior alveolar injection was given and testing resumed for another 5 minutes. The presence of subjective anesthesia (lip and tongue numb) but a positive response to either Green Endo-Ice® and/or electric pulp testing (less than 80/80) at the end of the testing cycle was assumed to indicate a lack of pulpal anesthesia and resulted in the patient receiving an intraosseous injection with the Stabident system. If the patient was negative to Green Endo-Ice® tests and achieved an 80/80 reading on the electric pulp tester, access of the pulp chamber was begun after placement of the rubber dam.

If the patient experienced any pain during access, but prior to exposure of the pulp, the patient received supplemental anesthesia via an intraosseous injection. Supplemental anesthesia was given via the intrapulpal injection, as described by Malamed (34), Walton and Torabinejad (14), and Cohen and Burns (92), when enough of the coronal pulp tissue

was accessible for a 27-gauge needle. This was done for expedience and it reflected what would occur in most clinical settings.

The intraosseous injections were given using the Stabident system with 1.8 cc of 2% lidocaine with 1:100,000 epinephrine. The selection of the Stabident system for this study was primarily to evaluate the system to see if it was effective and efficient in gaining clinical anesthesia as a supplementary injection on teeth diagnosed with irreversible pulpitis. The anesthetic solution of 2% lidocaine with 1:100,000 epinephrine was used because Replogle (28) found that the anesthetic success rate was lower and of shorter duration using a solution of 3% mepivacaine via the Stabident system.

The site of injection was designated as distal to the mandibular or maxillary test tooth, except if the test tooth was a second or third molar when it was given mesial to the teeth. The distal site is recommended in the Stabident manual (130) for mandibular teeth, but this has not been based on scientific study. The manual states that for maxillary posterior teeth either mesial or distal injections may be given with equal efficacy (again not proven). The distal site, and the mesial site for second and third molars, was chosen to maintain consistency with other studies (26,27,28) which have evaluated the efficacy of the intraosseous injection in experimental, mandibular first molars and adjacent teeth.

The site of osseous perforation was 2 mm below the horizontal line connecting the buccal gingival margins of the adjacent teeth and a vertical line passing through the interdental papilla in the attached gingiva (130). The site selection, as described by the Stabident Manual (130), allows the operator to inject apical to the thin, friable intercrestal bone and remain in attached gingiva which is less mobile. Bisecting the interdental papilla

means that the injection site will be equidistant between adjacent root structures. The mean width of attached gingiva, from the gingival margin to the mucogingival line, was reported by Bowers (166) to be 2.7 mm at the mandibular first molar, 2.2 mm at the mandibular second molar, 4.2 mm at the maxillary first molar, and 4.1 mm at the maxillary second molar. Ainamo et al. (167) reported results of 2.2 mm and 2.0 mm in mandibular first and second molar, and 2.6 mm and 2.5 mm for maxillary first and second molars, respectively. Tenenbaum and Tenenbaum (168) reported the mean width of attached gingiva to be 2.27±0.60 mm and 0.92±0.68 mm in mandibular first and second molars. First and second mandibular premolars averaged 1.12±0.57 mm and 1.69±0.61 mm, respectively. Maxillary first and second premolars averaged 1.98±0.90 mm and 2.72±0.89 mm, respectively, while first and second molars averaged 3.53±0.73 mm and 1.37±0.95 mm, respectively. This would suggest that the site of perforation is in attached gingiva. This was found to be true in the majority of cases in this study. In cases where it was not, the perforation site was moved coronal to the mucogingival line. Since attached gingiva is firmly bound to underlying periosteum and bone by fibrous attachment and since the collagen fibers of alveolar mucosa are fewer in number and less dense than in attached gingiva (58), the perforation site was easier to relocate for insertion of the needle.

All the subjects in this study had periodontal tissues considered to be within normal limits as determined by periodontal probing. It is currently unknown whether the selection of a site with periodontal disease, inadequate bony architecture, or periodontal pocketing would affect site selection or be a contraindication in using the Stabident system in the

area in question. Future studies could look at these parameters and determine alternative perforation sites.

No topical anesthetic was used prior to the infiltration of the mucosa in the area of the intraosseous injection. This is suggested by the Stabident Manual (130), however, we wished to measure the pain of the infiltration injection prior to the perforation. During intraosseous injection, patients rated the insertion of the needle and deposition of solution during local infiltration as well as the perforation and injection of anesthetic in the cancellous bone utilizing the 0-3 scale used for the nerve block and infiltration injections and utilizing the same hand signals. The rating of these parameters allowed for comparisons to be made to previous Stabident studies.

At the determined site of perforation, approximately 0.1 cc of 2% lidocaine with 1:100,000 epinephrine was deposited into the attached gingiva using a 27-gauge, ultra-short Stabident needle. This produced a light blanching of the tissue. Dunbar (26) found this volume to be adequate in 96% of his patients since they experienced no pain on perforation of the cortical bone. In this study, 16 of 24 (66.7%) patients felt no pain on perforation of the cortical plate and of the 8 that reported pain, 75% reported only mild discomfort. The Stabident Manual (130) recommends that if a patient still experiences sensitivity after injection in the attached gingiva, the operator can inject approximately 0.25 cc of anesthetic solution into the unattached gingiva and wait 30 seconds for diffusion. This was not done in this study so as to allow for the recording of pain ratings on perforation of the bone and injection of the anesthetic solution into the cancellous bone.

The Stabident perforator was placed in a contra-angle, slow-speed handpiece. The cap of the perforator was removed and the handpiece was activated to ensure the concentric rotation of the perforator. If the perforator was found to be severely out of round it was discarded and replaced with a new perforator. Severe eccentric rotation of the perforator may increase the chance of perforator separation in the bone. This did not occur in this study and may be related to the pre-testing of the perforators prior to their use.

The perforator was inserted into the hole created by the 27-gauge needle during infiltration of the attached gingiva. This helped prevent confusion in determining the perforation site by not having two holes in the tissue. The perforator was placed at a 90° angle to the cortical bone to allow for perforation along the shortest route through the bone. Any deviation of this angle may cause inadequate perforation due to the limited length of the perforator and an increase in the distance required to perforate the bone.

The handpiece and perforator were activated as they lightly contacted bone. Light pressure was applied until a feeling of "break through" was achieved. If this was not achieved within 2-5 seconds, the perforator was withdrawn still activated as recommended by the Stabident Manual (130). The "break through" feeling represents perforation into the cancellous bone. This was not always observed within the first 2 seconds of the first attempt. Reinsertion of the perforator in the same hole and reactivation of the handpiece was accomplished to complete perforation. Generally, failure was due to not attaining full depth of penetration with the perforator and can be attributed to varying thickness' of cortical bone. Denio et al. (169) have reported a mean thickness of cortical bone without

trabeculation to be between 2.7 mm and 3.0 mm between mandibular first and second molars. This is the area where all of the perforation difficulties were observed in this study. Cortical bone is generally thinner in the maxilla as compared to the mandible and the anterior regions are thinner than the posterior regions (170). The mean thickness of attached gingiva has been reported by Goaslind et al. (171) as 1.25 mm. Combining the above data reveals a mean thickness of between 3.95 mm and 4.25 mm that needs to be perforated. A pilot study conducted by Dunbar (26) determined that the mean length of the Stabident perforator was 8.4 mm with a range of 8.0 mm to 9.0 mm. This appears to be an adequate length to achieve perforation through the cortical bone and this was found to be true in this study.

If "break through" is not noticed and/or the perforator feels to have stopped and cannot be advanced any further then the perforator should be moved to another site. It is possible that the perforator was inserted at a wrong angle and may be hitting a root of one of the adjacent teeth at the site of injection. In a pilot study using pig mandibles, it was discovered that the perforator could drill into teeth. The amount of pressure required to do this was extremely high. The difference in feeling of perforating bone and drilling into a root is quite noticeable and was not a problem encountered in this study.

The perforator was removed from the bone while still activated to prevent breakage in the bone. The site of perforation was then identified by placing a cotton roll against the site and upon removal, a small dot of hemorrhage indicated the perforation site. Sometimes additional pressure was required due to heavy bleeding, but finding the perforation was not hindered.

A 27-gauge ultra-short Stabident needle on an aspirating syringe was then inserted into the perforation site. At times, due to the location and accessibility of the site, the needle had to be bent to approximately a 45° angle. The 27-gauge needle was found to be adequate in transversing the perforation and it was long enough to enter the cancellous bone. It was also found to be large enough to deliver the anesthetic solution and fit snugly in the perforation to prevent back-flow of the solution into the oral cavity.

Anesthetic solution, 1.8 cc 2% lidocaine with 1:100,000 epinephrine, was then delivered into the cancellous space over a 2 minute time period. The solution was delivered without back-pressure. In a few patients back-pressure was experienced initially and the anesthetic solution did not advance as readily into the cancellous bone. Rotation of the needle either clockwise or counterclockwise approximately a quarter turn was done and injection reattempted as suggested by the Stabident Manual (130). This was done to move the lumen of the needle away from the trabeculae of the cancellous bone, the periodontal ligament, the root, or the lamina dura which may have been blocking the deposition of the anesthetic. If this did not help, the needle was removed and checked for blockage by expressing some solution from the needle tip. In 3 of 24 patients (12.5%) back-pressure was felt during delivery of anesthetic. In all of the cases, reperforation of the initial site helped remedy the problem and the anesthetic solution was easily deposited.

The intraosseous injection technique consisted of the syringe being held in a "pen-gripping" fashion to allow for improved control of the needle in locating and entering the perforation site (130). It also gave the operator a better feel in locating, entering, inserting, and depositing the anesthetic solution into the cancellous bone.

All patients were asked to subjectively evaluate heart rate increases with the intraosseous injection during and for 2 minutes after the intraosseous injection. Responses of yes or no were recorded. Previous studies have reported that local anesthetic solutions containing epinephrine significantly increased the heart rate when injected intraosseously (26,27,28,54,55,56). The duration of effect was reported to be approximately 2-3 minutes at which time it diminished (28,54,55). The onset of an increased heart rate was reported by Lilienthal (55) to be within 10 seconds of the injection of a bolus of 0.9 cc of prilocaine with 1:200,000 epinephrine injected at a rate of 0.9cc/30 seconds. Replogle (28) reported that approximately 67% of patients registered a rise in cardiac rate (using an EKG monitor) during or 2 minutes following deposition of 2% lidocaine with 1:100,000 epinephrine using the Stabident intraosseous system. The mean rise in heart rate was reported to be about 22 beats from the normal rate. Replogle (28) also reported that 14% of patients actually had an increase of 37 beats in addition to a prolonged effect of 6 to 22 minutes.

In this study, the 1.8 cc of 2% lidocaine with 1:100,000 epinephrine was injected at a rate of 0.9cc/60 seconds intraosseously. This is based on the ease of injection and the ability to give the injection at this rate. Eleven of the 24 patients (45.8%) receiving the intraosseous injection reported a subjective feeling of heart rate increase in this study. Dunbar (26), Coggins (27), and Replogle (28) reported a combined rate of 75% of patients having a feeling of increased heart rate using the Stabident system and 1.8 cc of 2% lidocaine with 1:100,000 epinephrine.

After receiving the intaosseous injection, the test tooth was tested with both the electric pulp tester and Green Endo-Ice® in 1 minute sequential cycles for 3 minutes, as described previously in this section. A stopwatch was started immediately after completion of the intraosseous injection. Reports (26,27,28) have shown that onset of anesthesia is immediate for the Stabident intraosseous injection and testing for 3 minutes was done to confirm this. Patients who still responded to the electric pulp tester and/or the thermal test were given a second intraosseous injection with the Stabident system. This was either at another location (usually mesial to the tooth) or in the same area and a new perforation was performed. In 2 of 24 patients in this study, a second intraosseous injection was required. These procedures maximized the use of the Stabident intraosseous injection in gaining pulpal anesthesia.

Teeth responding negatively to both tests were isolated with a rubber dam and access of the pulp was started. Any pain during access, following the intraosseous injection, was recorded as well as the level of access, the postinjection time the pain occurred, and the pain rating (0-3) reported by the patient. If the pain occurred prior to full exposure of the pulp, a second intraosseous injection was given. If the pulp was exposed, intrapulpal anesthesia was given as described previously (14,34,92). Patients were asked to rate the pain perceived during supplementary intrapulpal injections (0-3) and this was recorded. Any pain experienced during endodontic treatment was recorded. The level of treatment, either accessing into dentin, exposing the pulp, removing pulp tissue, or filing the canals, was recorded as well as the pain as rated by the patient. These procedures duplicated

clinical endodontic treatment and the pain ratings were recorded to evaluate how painful these procedures would be clinically.

After completion of the emergency endodontic treatment and temporization of the tooth, patients were given prescriptions for pain medications as indicated and at the discretion of the operator. Patients were instructed to return to the clinic if any problems occurred, specifically, at the intraosseous injection site. Dunbar (26) reported only 1 of 80 patients (1.3%) had any postinjection complications which were related to the intraosseous injection. Coggins (27) reported a 2% incidence of postinjection problems. This included three exudative reactions which were treated with antibiotics and that cleared up within 7 days. In this study no patient returned or called with any complications related to the intraosseous injection.

Success of the intraosseous injection was defined as treatment that could be successfully completed without patient discomfort. Patients who experienced mild pain during access of an experimental tooth and then reported no pain upon receiving an intrapulpal injection were considered a success.

The results of the current study were placed in categories in order to evaluate the data statistically. These categories included the relationship of the electric pulp tester and Green Endo-Ice® to determine pulpal anesthesia, a comparison between the arches for teeth requiring intraosseous supplemental anesthesia, and the success/failure of the Stabident intraosseous injection in teeth diagnosed with irreversible pulpitis.

The statistical analysis of the correlation of the EPT to Green Endo-Ice® was done via the phi-coefficient. This was due to the nonparametric, nominal nature of the data. A

percentage of agreement and a kappa statistic were also determined. The comparison between the arches for teeth requiring intraosseous supplemental anesthesia was determined by the chi-square test. Again, this was due to the nonparametric, nominal nature of the data. The success of the Stabident intraosseous injection was determined by the Fisher Exact test. This test replaces the chi-square test when the sample size is too small. It too is a nonparametric, nominal test. Confidence intervals of 95% and 99% were also calculated in terms of mandibular success rates.

Results

Fifty-one subjects participated in this study. Table 1 shows the mean age of all the subjects to be 34.0 years with a range of 19 to 68 years. The 24 patients who received an intraosseous injection had a mean age of 32.6 years with a range of 19 to 63 years. While the effect of age on the ability to gain pulpal anesthesia has not been studied in adult patients, the two groups were balanced with respect to age. Therefore, if there would be a difference in anesthetic effect due to age, the balance between the two groups would minimize its effect on our results.

Twenty-three (45%) subjects were male and 28 (55%) subjects were female. Ten of 24 subjects (42%) who received an intraosseous injection were male while 14 of 24 (58%) were female (Table 1). Lautenbacher and Strian (172) found no sex differences in the perception of thermal sensitivity and pain to heat. Feine et al. (173) investigated sex differences in the perception of heat stimuli. They reported that females rated noxious heat stimuli as being more intense than males. They reasoned that the differences were probably related to sensory factors rather than attitudes or emotional differences. Lipman et al. (174) found no sex differences in terms of pain tolerance to noxious heat stimuli. There may be differences in the tolerance of heat induced pain between males and females. Any sex differences in the tolerance to pain were minimized in this study since there was a similar distribution of males and females in both the overall subject group and the group requiring the intraosseous injection.

The distribution of tooth type and arch is found in Table 2. Twenty-five teeth (49%) were maxillary and 26 of 51 (51%) were mandibular. Thirteen (25%) teeth were

premolars while 38 of 51 (75%) were molars. The largest group of teeth seen in this study were maxillary and mandibular first molars (47%). Posterior teeth were used exclusively in this study. Coggins (27) reported on the efficacy of the intraosseous injection using the Stabident system as a primary injection in normal maxillary and mandibular first molars and lateral incisors. He found the efficacy of the intraosseous injection decreased posterior to anterior in maxillary teeth and anterior to posterior in mandibular teeth. He also found that the Stabident injection was significantly more successful in maxillary first molars as compared to mandibular first molars. White et al. (21), using the periodontal ligament injection, found that the success of that intraosseous injection decreased from posterior to anterior. They reported no differences between maxillary and mandibular teeth. Therefore, based on these studies (21,27) it would be likely that the teeth used in this study would be anesthetized by an intraosseous injection. Walton and Abbott (20) reported that mandibular molars, maxillary and mandibular premolars, and maxillary molars, in that order, were the most difficult tooth groups to anesthetize, especially when diagnosed with irreversible pulpitis. Also, the number of posterior teeth requiring endodontic therapy generally outnumber anterior teeth in the specialty practice of endodontics. Therefore, only posterior teeth were used in this study since this distribution of teeth would represent the teeth most likely to be treated in an endodontic practice.

The initial mean pain intensity of all the teeth was 2.3 (Table 3). This correlated to pain that was between discomforting but bearable and pain that was discomforting and difficult to bear. This pain is representative of patients with an irreversible pulpitis (30)

who present for emergency treatment since they could not tolerate the pain any longer. Uhle (30), utilizing the same pain ratings as used in this study, reported a mean pain of 2.4 to 2.5 for patients diagnosed with irreversible pulpitis in a posterior tooth.

All of the patients responded to the electric pulp tester while 49 of 51 subjects responded to the thermal tests with Green Endo-Ice® (Table 3). None of the subjects had to be excluded from the study due to an incorrect diagnosis of irreversible pulpitis since all pulps were found to have vital coronal tissue upon access opening. The fact that 2 patients did not respond to the Green Endo-Ice® reflects that the status of the pulp cannot always be determined by clinical tests (88,109-113) and that inflammation of the pulp may be present with varying degrees of pain and sensitivity (92). It may also indicate that the Green Endo-Ice® was not sufficiently cold enough for a response in some patients.

Comparison of the pain caused by the electric pulp tester and the Green Endo-Ice® revealed that there was a significant difference ($p = 0.0039$) between the two (Table 3). The Green Endo-Ice® (mean pain rating of 2.6) caused more severe pain than the electric pulp tester (mean pain rating of 1.4) in the initial tests. This could be due to the regulated increase in electrical stimulation the patient received as the electric pulp tester ran through its cycle. A patient could react to the stimulus before it became too painful and thus would rate the pain associated with it as low. The Green Endo-Ice®, in contrast, was not controlled. A sudden and cold (-26°C) stimulus was applied to the tooth and caused a moderate to severe reaction. Patients with endodontically involved vital teeth may be sensitive to cold, hot and cold, or hot which is relieved by cold stimuli. Any of these responses may bring a patient into the office for treatment. These responses may be

reported as exaggerated and prolonged (30) and a similar response to cold stimulus may be elicited during initial Green Endo-Ice® testing. Augsburger and Peters (78) reported that skin refrigerant caused a mean decrease in intrapulpal temperature of 2.06°F within 5 seconds of application. Trowbridge et al. (175) reported that sensory responses to thermal stimulation in patients was actually faster than any change of temperature registered at the pulpodentinal junction. They theorized that hydrodynamic forces were produced by the temperature changes in the dentin and that these forces were the source of response within the tooth's sensory structures.

The mean pain rating caused by the Green Endo-Ice® (2.6) was higher than the initial mean pain rating (2.3) of the test tooth and was almost twice the value of the mean pain rating with the electric pulp tester (1.4). This would indicate that testing the vitality of a tooth, that a patient already has indicated as painful and sensitive to cold, with Green Endo-Ice® may cause undue pain to the patient as compared to the electric pulp tester. This may only be indicated in cases where the diagnosis of the offending tooth is required and/or when the electric pulp tester cannot be used. In this study, the cold test was conducted along with the electric pulp test to compare the accuracy of the two tests in confirming pulpal vitality and anesthesia.

Tables 4 through 6 summarize the pain ratings for the inferior alveolar nerve block and the maxillary infiltration for needle insertion, needle placement, and anesthetic solution deposition. The ratings were reported using the numerical scale previously used in studies at The Ohio State University (7,8,21,24,25,26,27,28,30,122,132-141,176,177).

Table 4 summarizes the pain reported on insertion of the needle 2-3 mm below the mucosa in both maxillary and mandibular injections. For mandibular nerve blocks, 20 patients (77%) reported no or mild pain while 6 of 26 (23%) patients reported moderate pain. For maxillary infiltrations, 23 of 25 (92%) patients reported no or mild pain and 2 of 25 (8%) reported moderate pain. Therefore, needle insertion has the potential to be moderately painful 8-23% of the time. There is a higher potential for moderate pain with the inferior alveolar nerve block as shown by Vreeland et al. (133), Nist et al. (136), and Goldberg (7) when compared to maxillary infiltration injections (Katz [177], Mason [143]). Even though topical anesthesia was used in this study, moderate pain was reported 8 to 23% of the time. The use of topical anesthetic has been advocated as an aid in reducing pain of needle insertion. However, even though Rosivack et al. (178) have shown the effectiveness of topical anesthetic, studies by Nist et al. (136), Yonchak (137), Mason (143), and Gill and Orr (179) have not. The most important effect of using topical anesthetic may not be its clinical effectiveness, but rather the psychological effect on the patient who feels the practitioner is doing everything possible to prevent discomfort.

The results of pain on needle insertion fall within a wide range of needle insertion pain ratings recorded in other studies. Katz (177) and Mason (143) reported moderate to severe pain on needle insertion in 3%-10% of maxillary infiltration injections. Gross (176) reported moderate to severe pain in 30-38% of the patients receiving a maxillary infiltration for the first molar. Needle insertion for inferior alveolar nerve blocks was reported to cause moderate to severe pain in 12% of the injections as reported by Hinkley (135), 26% by Nist et al. (136) and Goldberg (7), and 37% by Vreeland et al. (133).

These differences can be accounted for by variations in each operator's technique, differences in subject pool (symptomatic versus asymptomatic patients), differences in the definition of insertion, and the use of topical anesthetic or a placebo prior to needle insertion.

Pain ratings for needle placement for both the maxillary and mandibular injections are found in Table 5. For maxillary teeth, all 25 patients (100%) reported mild or no pain on placement of the needle to the target site for the injection. For mandibular teeth, 17 of 26 (65%) of patients reported mild or no pain and 9 of 26 (35%) reported moderate to severe pain. The increase in the number of moderate to severe pain ratings during needle placement in mandibular injections could be due to the structures (connective tissue and muscle tissue) that need to be penetrated or traversed to reach the target site and possible trauma to the periosteum on contact of the needle with bone (34).

For inferior alveolar nerve blocks, Dunbar (26) reported that 24% of patients recorded moderate to severe pain on placement of the needle to the target site. Childers (122) reported 35% and Clark (138) reported 63% of patients recording a moderate to severe response. Again, these differences could be due to operator differences and differences in the types of subjects utilized (symptomatic or asymptomatic) in the studies. For maxillary infiltrations, no other study divided the injection into three distinct categories as this one did for posterior teeth. Katz (177), Mason (143), and Gross (176) considered needle insertion and placement as one stage and reported pain ratings as such (see above discussion).

The pain ratings for solution deposition, in the present study, are found in Table 6. In mandibular nerve blocks, 8 of 26 (31%) patients recorded moderate to severe pain. For maxillary infiltrations, 2 of 25 (8%) injections elicited moderate pain. The results of other studies, utilizing the inferior alveolar nerve block, reported incidences of moderate to severe pain for solution deposition as follows: Dunbar (26) - 21.3%; Childers (122) - 17.5%; Nist et al. (136) - 25%; Goldberg (7) - 13.3%; Hinkley (135) - 11.1%; Wali (132) - 14.4%; and Vreeland et al. (133) - 36.7%. For posterior maxillary teeth, using an infiltration injection of 2% lidocaine with 1:100,000 epinephrine, Gross (176) reported 39%, Katz (177) - 13%, and Mason (143) reported 7% moderate to severe pain on solution deposition. Variations in operator technique, patient population, quantity of anesthetic deposited, rate of anesthetic solution deposition, use of symptomatic or asymptomatic teeth, and neural anatomy in the injected areas could all account for the differences in pain ratings for these injections.

There was a 0% incidence of positive aspiration during the injections. Malamed (34) reported an expected incidence of positive aspiration during the inferior alveolar nerve block of 10-15%. He also reported an expected positive aspiration rate for maxillary infiltrations of less than 1%. Clark (138) reported a 14.4% incidence of positive aspirations with the inferior alveolar nerve block. Nist et al. (136) reported an incidence of 27.5% and Clark (141) and Childers (122) both reported an incidence of 0%. The differences in this study and the others was probably due to the definition of positive aspirations. We defined this as aspiration of a relatively large amount of blood; whereas, others have defined this as the presence of any blood within the cartridge.

Subjective lip anesthesia was obtained in 19 of 26 (73%) of the initial inferior alveolar nerve blocks. Seven patients required a second block injection due to failure in gaining subjective lip numbness after 5 minutes of testing. These failures were most likely due to anatomic variation between patients (34). Most experienced clinicians will have a low incidence of missed inferior alveolar nerve blocks, but they do occur. Dunbar (26) reported a 11.1% failure rate after the initial injection failed to gain subjective lip anesthesia. Childers (122) reported a 15.9% incidence of "missed" blocks. Clark (138) reported a 6.2% incidence, Clark (141) a 4.0% incidence and Hannan (140) and Simon (139) an incidence of 10.9% and 8.5%, respectively. The incidence of "missed" inferior alveolar nerve blocks in this study could be due to the time constraint of a 5 minute waiting period. Previous studies (7,133) have shown that the onset of lip anesthesia may occur after 5 minutes. Therefore, waiting longer than 5 minutes could have reduced the number of subjects requiring a second inferior alveolar injection in this study. Although lip anesthesia demonstrates a "clinically" successful inferior nerve block, it does not guarantee the onset or success of pulpal anesthesia (7,26,122,132,133,135,136). All 7 patients requiring a second block injection gained signs of subjective lip numbness and thus 100% of the mandibular cases did have signs of a "clinically" successful inferior alveolar nerve block before any further treatment was rendered.

No subjective signs of anesthesia were used for the maxillary patients. The relatively high rate of success of the maxillary infiltration using 3.6 ml of 2% lidocaine with 1:100,000 epinephrine, over 95% (34), would indicate that this injection would be successful even though no subjective signs could be used to determine infiltration success.

Table 7 summarizes the results of testing with the Analytic Technology® electric pulp tester and Green Endo-Ice® after anesthesia was given. At minute 1, 19% (5 of 26) of the mandibular teeth tested 80 with the electric pulp tester while 42% (11 of 26) of the teeth responded negatively to Green Endo-Ice®. The difference between the two negative responses may indicate that thermal sensitivity may be lost sooner than electrical reactivity or proprioception as reported by Reeve (39). However, the difference may also be related to the temperature of the Green Endo-Ice®. At minute 2, 35% (9 of 26) of the mandibular teeth gave an 80 reading and 54% (14 of 26) tested negative to Green Endo-Ice®. In each case, the number of negative responses increased. At minute 3, 35% (9 of 26) of the mandibular teeth still gave an 80 reading and 69% (18 of 26) were negative to thermal testing. This reveals that the reaction to cold continued to decrease while reaction to the electric pulp tester was similar to the 2 minute time period. Forty-two percent (11 of 26) of the mandibular teeth gave an 80 reading and 81% (21 of 26) tested negative to Green Endo-Ice® at minute 4, and at the end of 5 minutes, only 12 of 26 (46%) teeth recorded an 80 reading with the electric pulp tester and 21 of 26 (81%) tested negative to Green Endo-Ice®. Overall, one can see that as the testing time increased so did the percentage of negative responses to both the electric pulp tester and the Green Endo-Ice®, and hence, the onset of pulpal anesthesia. Testing for a longer time period may have increased the percentage of negative responses since it would have included more of the patients experiencing slow onset of anesthesia. Dunbar (26) reported a range of slow onset (80 reading after 20 minutes) of 10-22.5% in mandibular posterior teeth. Childers (122) reported an incidence of 10-15%, Hannan (140) an incidence of 17.5-25%, Simon (139)

an incidence of 33-45%, and Wali (132) an incidence of 47-67% of slow onset for mandibular posterior teeth.

In maxillary teeth, Table 7 shows that at minute 1, 60% (15 of 25) of the teeth gave an 80 reading and 84% (21 of 25) responded negatively to Green Endo-Ice®. These percentages are much higher than those achieved in mandibular teeth at the same time period and may indicate faster onset of pulpal anesthesia in maxillary teeth. Again, the percentage of teeth responding negatively to the Green Endo-Ice® was higher versus the electric pulp tester. This too may indicate that thermal sensation is lost sooner than electrical reaction after local anesthesia is given or these readings may be related to the temperature of the Green Endo-Ice®. At minute 2, the gap between the two test percentages narrowed. At minute 3, both tests resulted in a 92% (23 of 25) negative response. These results indicate that, with time, the onset of pulpal anesthesia in maxillary teeth increases and that by 3 minutes, 92% of the patients have pulpal anesthesia when tested with either the electric pulp tester or Green Endo-Ice®. Testing for a longer time period may have increased the percentage of negative responses due to patients experiencing slow onset of anesthesia (80 reading after 7 minutes). Mikesell (142) reported a 9% incidence of slow onset in maxillary molars. Gross (176) reported a 34% incidence, Mason (143) an 11% rate, and Katz (177) a 10% incidence of slow onset of anesthesia in maxillary first molars.

Combining the end of testing results revealed that 35 of 51 (67%) teeth recorded an 80/80 with the electric pulp tester and 44 of 51 (86%) responded negatively to the Green Endo-Ice® (Table 7). Statistical analysis of this data revealed a moderate level of

agreement between the two tests (phi coefficient = 0.648) in determining pulpal anesthesia (Table 8). There was an 80.39% agreement rate between the two tests with a kappa value of 0.617. This too indicates a moderate level of agreement between the two testing procedures. However, when looking at the results in Table 7 in terms of separate arches, individual differences are seen. In the mandible, the percentage of 80 readings with the electric pulp tester was always lower than the percentages of negative responses to Green Endo-Ice®. If, in this study, we had only used the Green Endo-Ice® as a testing criteria, only 5 (19%) of the cases would have received supplemental anesthesia prior to endodontic treatment. If the electric pulp tester had been used solely, 14 (54%) of the cases would have received supplemental anesthesia. When looking at research on the success rates of the inferior alveolar nerve block in asymptomatic teeth, the success has been reported to be approximately 55-58% (11,132-139,141). This is certainly closer to the result achieved in this study using the electric pulp tester (46%) as compared to the Green Endo-Ice® (81%). If we also consider the fact that symptomatic teeth have been reported to be more difficult to anesthetize clinically (150), the percentage of success would be expected to be closer to the results with the electric pulp tester. It then may be conjectured that the electric pulp tester is more accurate in determining pulpal anesthesia in symptomatic, mandibular posterior teeth than Green Endo-Ice® and that if the two tests are run and opposing results occur, the electric pulp tester result may be the most valid.

In maxillary teeth, the percentages of negative responses to Green Endo-Ice® were higher than the percentages of 80 readings for the first 2 minutes of testing (Table 7). They were the same after minute 3. This may indicate that the onset of pulpal anesthesia

occurs between minute 2 and minute 3 in maxillary teeth. This would confirm the results of Mikesell (142) who reported a 2.42 minute onset rate in maxillary first molars using 3.6 ml of 2% lidocaine with 1:100,000 epinephrine. The incidence of first molar anesthesia, using 3.6 ml of 2% lidocaine with 1:100,000 epinephrine, has been reported to be 78% at 3 minutes (142). Our results had a higher success rate (92%) at 3 minutes (Table 7) and this may have been the result of variations in the subject population or the smaller number of teeth used in this study. Therefore, in contrast to the lower success rate in the mandible, maxillary infiltrations would be more successful at 3 minutes. We may then conclude that the electric pulp tester and the Green Endo-Ice® may be equally effective in determining pulpal anesthesia in maxillary posterior teeth.

Anesthetic success, in this study, was defined as an 80/80 reading with the electric pulp tester and a negative response to tests with the Green Endo-Ice® at the end of the testing time period. This definition differs from previous studies (7,24,25,26,27,28,122,29,132-144) in that they defined success as two consecutive readings of 80/80. Since this study was clinically applied, it was decided that tests after the prescribed time would define the next course of treatment by the clinician, either give supplemental anesthesia or begin endodontic therapy. This decision may have effected our results since we included patients who had slow onset of anesthesia and who may not have required supplemental anesthesia if additional time was given for anesthetic onset. We also may have included patients whom were having noncontinuous anesthesia in that these patients may have recorded 80/80 and were negative to Green Endo-Ice® at the last testing period, but who would have responded to either test at later testing intervals. The success rates of the

initial injections may also have possibly been higher if we had tested for a longer time interval for the onset of anesthesia; but it could have been possibly lower if we considered subjects having noncontinuous anesthesia. The clinician needs to be aware of the conditions of slow onset and noncontinuous anesthesia and the effects these conditions have on the results of both the electric pulp and Green Endo-Ice® test times.

Noncontinuous anesthesia may occur due to a fluctuation between the free base form and the cationic form of the local anesthetic at the sodium channel of the nerve membrane. This fluctuation in the dynamic equilibrium may lead to the blocking and then the unblocking of the sodium channels and thus the noncontinuous anesthesia. Dunbar (26) reported an incidence of 22.5-27.5% noncontinuous anesthesia in mandibular posterior teeth after an inferior alveolar nerve block. Childers (122) reported a rate of noncontinuous anesthesia of 20-25%. Hannan (140) recorded a rate of 12.5-25% while Wali (132) reported rates of 10-16% in mandibular posterior teeth.

Since the onset of lip anesthesia generally occurs within 5 minutes for the inferior alveolar nerve block (7,26,122,132-136,138,141) and the onset of pulpal anesthesia occurs within 3 minutes for maxillary posterior teeth when using 3.6 ml of 2% lidocaine with 1:100,000 epinephrine (142), this study was designed to utilize these times since, clinically, most practitioners would wait the prescribed times and then decide the next course of treatment depending on the test results.

The percentage of anesthetic success, 46% (Table 7), utilizing the Analytic Technology® electric pulp tester as a diagnostic test for mandibular posterior teeth is comparable to the following results: Dunbar (26) - 37.5-45%; Childers (122) - 60-72.5%;

Nist et al. (136) - 43-53%; Hannan (140) - 57.5-60%; and Clark (138) - 47-73%. In these studies, electric pulp tests were run on non-inflamed second premolars and first and second molars. Using symptomatic teeth, we would expect an effect on the success rate of the inferior alveolar nerve block (11). Wallace et al. (150) reported that endodontists did find symptomatic teeth more difficult to anesthetize than asymptomatic teeth. The percentage of negative responses with Green Endo-Ice® (81%) in mandibular posterior teeth diagnosed with irreversible pulpitis was higher than the 63% reported by Cohen et al. (17). However, in his study he utilized Frigi-Dent (dichlorodifluoromethane) which has a temperature of -50°C (17) compared to Green Endo-Ice® (tetrafluoroethane) which has a temperature of only -26.2°C (89). The differences in these temperatures could explain why more patients responded to thermal testing in Cohen's study. Green Endo-Ice® has a temperature only slightly colder than ethyl chloride. Fuss et al. (16) reported that ethyl chloride was inferior to dichlorodifluoromethane and the electric pulp tester in determining pulpal vitality. We may conclude from our results that Green Endo-Ice® may not be as accurate as dichlorodifluormethane nor the electric pulp tester in determining anesthesia and that it may be closer to ethyl chloride in reliability of determining anesthesia in mandibular teeth.

Success of anesthesia for the maxillary posterior teeth was 92% utilizing the electric pulp tester (Table 7). Mikesell (142) reported an incidence of anesthetic success of 78% using 3.6 ml of 2% lidocaine with 1:100,000 epinephrine in noninflamed maxillary first molars. Dreven (11,29) reported a success rate of 97% using 3.6 ml of 2% lidocaine with 1:100,000 epinephrine in noninflamed maxillary teeth. Again, the definition of success in

those studies differed than in this current study, but they do show that maxillary infiltrations are more successful than mandibular nerve block anesthesia (7,26, 122,132-138,140,141). This study may also confirm the contention of Mikesell (142) that 3.6 ml of 2% lidocaine with 1:100,000 epinephrine is more efficacious than 1.8 ml of the same anesthetic although no direct comparison was made in this study.

The test utilizing the Green Endo-Ice® gave an identical result (92%) as the electric pulp tester in maxillary teeth. No other studies have reported on the use of Green Endo-Ice® in determining pulpal anesthesia in maxillary teeth. Fuss et al. (16) reported that the electric pulp tester and dichlorodifluoromethane (DDM) were reliable in determining pulpal vitality in uninflamed premolars. However, the group did not establish whether the DDM was reliable in determining pulpal anesthesia to the level required by endodontists. From our results, Green Endo-Ice® appears to be as reliable as the electric pulp tester in determining pulpal vitality in symptomatic, maxillary posterior teeth. If both tests are run on these teeth and opposing results are achieved, the practitioner may have to follow the test results he/she feels more confidence with. Since the electric pulp tester has been proven to be a valid indicator in determining pulpal vitality (2,11,16,29,69,72) we feel that its results carry more weight.

The pain related to pulp testing with both the electric pulp tester and the Green Endo-Ice® is found in Table 9. At the first minute of testing, 1 patient rated the pain caused by the electric pulp tester as moderate and 3 patients rated the pain caused by the Green Endo-Ice® as moderate to severe. At minute 2, only 1 patient rated the pain due to testing with the Green Endo-Ice® as severe while no patient rated the pain from electrical pulp

testing as higher than mild. At minute 3 and all times after that, neither testing modality was rated as moderate or severely painful. This is most likely due to the onset of pulpal anesthesia to some degree. The average time of onset for mandibular pulpal anesthesia has been reported to be approximately 16 minutes (7,26,132-138,141), which includes patients with slow onset of anesthesia. The mean onset time for pulpal anesthesia in maxillary teeth has been reported as 5-7 minutes (142,143,144,176,177). All of these studies utilized the electric pulp tester as the means of testing pulpal vitality, but many of these studies utilized test times of 2 and 3 minute intervals as compared to the 1 minute intervals used in this study. We concluded that testing with Green Endo-Ice® at 1 minute postinjection time may be more painful than testing with the electric pulp tester. However, if the practitioner was to wait a reasonable length of time postinjection (for mandibular teeth - 5 minutes, maxillary teeth - 3 minutes) before testing, the pain ratings caused by either test would be mild. The practitioner also would gain more clinically usable test results when testing later than 1-2 minutes postinjection.

At the end of the testing period, 5 minutes for mandibular teeth and 3 minutes for maxillary teeth, determination of the next step in treatment was made. A total of 18 of 51 (35%) subjects in this study required an intraosseous injection due to positive responses to the electric pulp tester and/or Green Endo-Ice® (Table 10). Dreven (11,29) reported that 3 of 30 (10%) subjects never recorded an 80/80 with the electric pulp tester with teeth diagnosed as having irreversible pulpitis. However, in his study he tested for 10 minutes before determining whether a supplemental periodontal ligament injection was to be given. He also included anterior teeth in his study. These two differences as well as the smaller

sample size may explain the differences in results. Cohen et al. (17) reported that 38% (23 of 61) of mandibular first molars diagnosed with irreversible pulpitis tested positive with DDM after a clinically successful inferior alveolar nerve block. These 23 teeth received periodontal ligament injections prior to endodontic treatment. In this study, 16 of 26 (61%) mandibular teeth required intraosseous anesthesia due to positive responses to either the electric pulp tester and/or the Green Endo-Ice® after a clinically successful inferior alveolar nerve block (Table 10). Breaking this group into categories resulted in 11 of 26 (42%) mandibular teeth which were positive only to the electric pulp tester, 2 of 26 (7%) were positive only to the Green Endo-Ice®, and 3 of 26 (11%) were positive to both tests (Table 10). Dreven (11,29) reported that 67% (2 of 3) of the failures he had in gaining a negative electric pulp test response were mandibular teeth. Cohen et al. (17) had a 38% failure rate in gaining a negative response to the DDM in mandibular first molars. In their study they utilized both 2% lidocaine with 1:100,000 epinephrine and 3% mepivacaine as compared to our study where we used only 2% lidocaine with 1:100,000 epinephrine. However, the use of 3% mepivacaine should not have affected the anesthetic success rates since McLean et al. (134) and Cohen et al. (17) found that there was no difference between 2% lidocaine with 1:100,000 epinephrine and 3% mepivacaine in achieving pulpal anesthesia in mandibular molars. Two of the 25 (8%) maxillary teeth responded positive to both tests (Table 10). These too required intraosseous anesthesia because they responded to both the electric pulp tester and the Green Endo-Ice® after maxillary infiltration injections (Table 10). Dreven (11,29) reported that 1 of 3 (33%) of his failures in gaining a negative reading with the electric pulp tester was a maxillary

molar. Differences in the results are due to the large discrepancy in the subject numbers between the two studies. However, from the results of this study and Cohen et al. (17) we can conclude that mandibular posterior teeth diagnosed as having irreversible pulpitis have the potential to not be anesthetized after achieving a clinically successful inferior alveolar nerve block.

Difficulty in gaining pulpal anesthesia in teeth with inflamed pulps has been widely reported (11,13,14,17,29,34,92,100,180). Initially it was thought that inflammatory changes reduced the pH of local tissues and thus reduced the ability of anesthetic solutions to penetrate nerve membranes (92). This explanation, however, does not explain why a nerve block, at a site distant from the inflammatory changes, may not produce pulpal anesthesia even when all the subjective signs are present.

The effects of tissue damage due to inflammation may help explain the difficulty of gaining complete anesthesia. Hudson (181) reported that inflammatory products may interfere with the absorption of anesthetic agents. Najjar (182) reported finding degeneration products at nerve sites distant from the actual site of tissue inflammation. He stated that these products may interfere with the nerve membrane's properties, or the action of local anesthetics. Wallace et al. (150) reported finding periapical nerve membrane changes after intentional pulp tissue damage in cats. They also found that impulses could be conducted through an area of nerve bathed with 2% lidocaine with 1:100,000 epinephrine when an inflamed pulp is stimulated. The group postulated that the membrane changes may have blocked the ability of the anesthetic solution to dissociate across the membrane and thus prevent anesthesia. The inflammation may also cause

nerves in the region to have altered resting potentials and excitability thresholds along the whole nerve fiber and that these changes allow for a flow of ions and action potentials even under conditions of "anesthesia." Mumford (183) suspected that clinical dental anesthetic blocks would not abolish all neural activity in a particular nerve and that increases in neural activity would create a greater possibility of neural transmission. Rood (184) reported that neural responses would increase after thermal injury due to inflammation of the surrounding areas of skin. He felt that this increase in the number of responses was due to nerves being sensitized by the conditions associated with inflammation. Brown (185) reported that compound action potentials increased when a nerve was put in contact with inflammatory exudate. He concluded that these inflammatory products would increase nerve activity.

Those teeth that tested negative to both the electric pulp tester and the Green Endo-Ice® were isolated with a rubber dam and endodontic therapy was initiated. Fourteen of 33 patients (42%) reported pain during treatment and required supplemental anesthesia (Table 11). This consisted of 6 of 23 (26%) maxillary teeth and 8 of 10 (80%) mandibular teeth. Dreven (11,29) reported a rate of 27% anesthetic failure in patients with symptomatic teeth and who tested 80/80 with the electric pulp tester. Cohen et. al. (17) reported that 4 of 34 (12%) patients who initially tested negative to DDM required supplemental anesthesia due to pain on performing a pulpotomy and that their overall diagnostic success rate using only DDM was 92%. The differences in results could be due to a number of factors. Dreven (11,29) included maxillary incisors in his study and none of these teeth (4) failed to gain total pulpal anesthesia and thus increased his rate of

success. He also tested for 10 minutes prior to beginning treatment and may have thus reduced the number of slow onset subjects in his study. Cohen et. al. (17) waited for their patients to report complete lip numbness or at least 5 minutes, and sometimes longer, before testing with DDM. By doing this, they may have reduced the number of failures by reducing the number of patients having slow onset of anesthesia. Although not directly compared, the use of DDM in determining pulpal anesthesia in symptomatic teeth, as used by Cohen et al. (17), may be more reliable than Green Endo-Ice®. When comparing DDM to the electric pulp tester in determining pulpal anesthesia, no differences in results can be seen. The results show that the initial percentages of patients not being numb after the inferior alveolar nerve block were similar using the electric pulp tester in our study and DDM in Cohen's study. Direct comparison of the two testing modalities is impossible since DDM is no longer available, on the market, due to it being classified as an environmental hazard. Differences in the number of patients reporting pain after testing negative to the electric pulp tester and Green Endo-Ice® or DDM between this study and Cohen et al. (17) could be due to differences in patient population. In our study, emergency patients, many of whom had no general dentist of record, came to our public clinic because they were in severe pain and were only interested in relieving that pain. Cohen et al. (17) studied patients in a private endodontic practice in which most patients are referred from a general dentist. These patients, generally, have a high dental IQ and have previous experiences with dental and even endodontic treatment. These differences in dental background and expectations may have led more of our patients to report pain since their level of anxiety was very high and they had very few previous dental

experiences. Also, in our study we wanted patients to report even the slightest discomfort they experienced during treatment so we could study pain as related to anesthesia. Cohen et al.'s (17) study did not focus on patient pain ratings and therefore may not have had patients report it. This may have led to more of our patients reporting pain or discomfort even though it was tolerable for them and they may not have normally mentioned it.

Five of the 33 subjects (15%) that responded negative to both the electric pulp tester and Green Endo-Ice® reported pain when dentin was accessed (Table 12). One of these teeth was in the maxilla while the other 4 were mandibular teeth. Dreven (29) reported that 1 of his 8 failures (teeth that had recorded an 80/80 reading with the electric pulp tester, but had pain during treatment) was due to pain when accessing dentin. This failure was in a maxillary tooth. Cohen et al. (17) did not report on the level of access when any of their 4 mandibular molar subjects reported pain. In this study, the 5 subjects who experienced pain when the dentin was accessed all received intraosseous injections (Table 12). We can conclude from our results that as many as 15% of patients, recording negative responses with the electric pulp tester and Green Endo-Ice®, may experience pain when the dentin of a symptomatic tooth is accessed.

Nine of 33 teeth (27%) that recorded an 80/80 with the electric pulp tester and a negative response with the Green Endo-Ice® had pain when the pulp was accessed (Table 13). Five of these teeth were maxillary and 4 were mandibular teeth. Three subjects (9%) reported pain when the pulp chamber was accessed, 5 (15%) when the pulp was removed, and 1 (3%) during filing of the canals (Table 13). Eight of these nine patients received

intrapulpal injections. One patient received an intraosseous injection because the pulp exposure was so small that a needle could not be inserted into the opening (Table 13). Dreven (29) reported that 7 of 8 teeth that tested negative to the electric pulp tester had pain when the pulp was instrumented. Six of these teeth were mandibular posterior teeth and 1 was a maxillary premolar. Cohen et al. (17), again, did not report the stage of treatment their 4 teeth (all mandibular first molars which tested negative to DDM) were at when pain was reported. However, it may be inferred that the pain occurred when the pulp tissue was exposed since they reported that these teeth received intrapulpal injections. From our results we can conclude that even when symptomatic posterior teeth test negative to the electric pulp tester and Green Endo-Ice®, pain may be experienced by the patient when the pulp is accessed in 27% of cases and that this can occur in either arch.

A possible explanation for the lack of pulpal anesthesia after a negative response to the electric pulp tester may be that the Analytic Technology® electric pulp tester, with a maximum current output of 50 microamps, is an insufficient stimulus. Bjorn (2) utilized an electric pulp tester with a maximum current output of 140 microamps and reported that teeth that did not respond to this output required endodontic therapy. He did not, however, test pulpal anesthesia. The maximum output level of 50 microamps was recommended for diagnostic use by Pepper and Smith (154), and Matthews et al. (65). Both groups believed that all normal teeth would respond to 50 microamps. Mumford (186) reported that different histopathologic conditions of the pulp would not raise or lower the electric pulp tester threshold. Therefore, all teeth that are vital should respond

to 50 microamps. Unfortunately the relationship between the diagnostic and anesthetic threshold is not known. Perhaps a stronger output is required to test pulpal anesthesia in teeth with irreversible pulpitis. Bjorn (10) reported that the peridental nerves would be stimulated at 200-400 microamps. The unit he developed had an output of 140 microamps and he reported that it did not elicit peridental or false positive readings in terms of vitality testing. Perhaps increasing the output of the Analytic Technology® electric pulp tester, within 140 microamps, would help in identifying teeth that may require supplemental anesthesia. Further research may resolve this question. There are electric pulp testers available that do produce a stronger stimulus than 50 microamps, however Matthews and Searle (187) have reported that they all have other electrical design deficiencies.

The types of nerve fibers being stimulated by the electric pulp tester or thermal tests may explain why diagnostic tests for pulpal anesthesia are not 100% reliable. Trowbridge (180) stated that C-fibers are stimulated by tissue damage while Aδ-fibers are concerned with immediate protective responses to noxious stimuli not related to tissue damage. Physiologic studies (188,189,190) have reported that electrical stimulation of teeth is due to Aδ nerve stimulation. If this is true, then C-fiber anesthesia may not be tested with the electric pulp tester. Achieving an 80 reading and being able to work on a tooth without eliciting pain would mean that both the Aδ- and C-fibers are anesthetized. If not, perhaps either the C-fibers are not anesthetized or our stimulus is not great enough to determine Aδ anesthesia. If the electric pulp tester tests both Aδ- and C-fibers and an 80 reading is achieved, but there is pain on entering the pulp, then our stimulus may be inadequate. If

pulpal inflammation causes changes in C-fibers and they are not stimulated by the electric pulp tests, then there is a potential for pain due to the altered nerves and the inability of the anesthetic agent to block conduction of impulses.

The afferent nerve fibers for temperature are A- and C-fibers (59). Trowbridge et al. (175) conjectured that initial sensory responses due to thermal changes are not actually due to changes in pulpal temperature, but rather due to physical changes in the dentin which secondarily produce excitation of the sensory fibers. This group postulated that cooling dentin would cause contraction and produce narrowing of the dentinal tubules, thus causing hydrodynamic forces which may decrease fluid volume within the dentinal tubules. This in turn would distort nerve fibers at the pulpodentinal junction. It is the A-fibers that are responsible for mechanical sensations (59) and the C-fibers may never actually be tested for anesthesia with thermal tests nor with the electric pulp tests. Along with the Aδ-fibers and C-fibers, Aβ-fibers have also been described in pulpal tissue (191). Aβ-fibers have been shown to carry pain sensations when a state of inflammation is present in cutaneous tissues and C-fibers have been activated (192). Since Aβ-fibers are large myelinated fibers, inflammatory changes may also alter their membranes and prevent anesthetic solution penetration.

All of these theories are conjecture. Electric pulp testing and Green Endo-Ice® testing may give the practitioner a good idea as to the presence of pulpal anesthesia, but it is not until the direct manipulation of the pulp tissue that the true state of anesthesia can be assessed.

Twenty-four of 51 (47%) patients in this study received an intraosseous injection utilizing the Stabident system while 8 of 51 (16%) patients required intrapulpal anesthesia (Table 14). For those requiring intraosseous anesthesia, 21 of 26 (81%) were mandibular teeth and 3 of 25 (12%) were maxillary teeth (Table 14). Statistical analysis revealed that a significant difference ($p = 0.0000$) for supplemental anesthesia between the two arches existed (Table 15). This result coincides with other reports that have found mandibular teeth to be more difficult to adequately anesthetize (3,14,20,92).

The problem of failing to achieve pulpal anesthesia in mandibular teeth has often implicated the inferior alveolar nerve block. Some explanations for the failure include accessory innervation, the central core theory, or impenetrable fascial planes.

The mylohyoid nerve has been implicated as a main source of accessory innervation to the mandibular teeth (34,63,193,194). Frommer et al. (63) showed that the mylohyoid did contain both sensory and motor nerves and that the sensory nerves were given off in the posterior region of the mandible. However, innervation of the teeth was not proven. Wilson et al. (193) and Madeira et al. (194) showed that the mylohyoid nerve extended to foramina at, and entered, the lingual aspect of the anterior mandible. Clark (138) showed that the mylohyoid nerve did not contribute any significant accessory innervation to the mandibular teeth. Therefore, based on Clark's work, it seems unlikely that the mylohyoid nerve makes any significant contribution to mandibular teeth.

The central core theory (195) states that nerve fibers which supply more terminally located structures are located centrally in a nerve trunk and that proximal structures are located peripherally on the nerve trunk. This means that posterior teeth should have a

higher rate of anesthetic success since their fibers are located on the periphery of the nerve trunk and the anesthetic solution should readily reach them. The results of Goldberg (7), Wali (132), Vreeland et al. (133), Hinkley (135), Nist et al. (136), and Clark (138) somewhat support the theory. All reported a higher incidence of anesthetic success in mandibular molars than lateral incisors, but not in premolars, using the inferior alveolar nerve block. Hannan (140) and McLean (134) showed that the onset of anesthesia occurred first in molars, then premolars and then in anterior teeth. This supports the central core theory also. Simon (139) and Hinkley et al. (135), however, showed faster onset of anesthesia with premolars followed by the molars and then anterior teeth. The central core theory may explain slow onset of anesthesia and why some mandibular teeth never gain anesthesia.

Fascial planes may limit the spread of anesthetic solution within the pterygomandibular space. Barker and Davies (196) and Berns and Sadove (197) have described the fascia that surrounds the inferior alveolar neurovascular bundle and how it effects the distribution of solutions within the pterygomandibular space. Berns and Sadove (196) have suggested that the closer the injection is to the nerve, the greater the chance for successful anesthesia. Simon (139) and Hannan (140) used a peripheral nerve stimulator and ultrasonic imaging, respectively, to deliver very precise injections. They found that their success rate was no greater than giving the inferior alveolar nerve block in the conventional way. Therefore, accuracy of the injection may not be a reason for failure.

The intraosseous injection may address some of the problems associated with anesthetic failure. Since the anesthetic solution is deposited into the cancellous bone

surrounding the roots of teeth, all the nerves, no matter the origin, should be anesthetized as they enter the root. Also, the anesthetic solution does not need to penetrate a large nerve trunk since its site of action is at the terminal branch of the nerve where the fiber has its smallest diameter.

Tables 16 and 17 summarize the pain ratings reported by subjects on access of teeth that had recorded 80/80 with the electric pulp tester and were negative to the Green Endo-Ice® tests. Ten of 14 (71%) reported moderate to severe pain. Thirty-six percent of these patients experienced their pain when dentin was reached by the high-speed handpiece (Table 16). Four patients reported the pain as moderate and 1 as mild. Dreven (29) reported that one patient experienced pain when dentin was instrumented (after recording an 80/80 with the electric pulp tester in a symptomatic tooth) and it was described as severely painful. Cohen et al. (17) did not record pain ratings reported by patients during treatment. In our study, these patients received intraosseous injections since intrapulpal injections were not feasible. Six of 14 patients (43%) reported moderate to severe pain when the pulp was exposed or removed. Three of 14 patients (21%) reported mild pain during exposure or removal of the pulp and during filing. Dreven (29) reported that 7 of 8 (88%) patients had severe pain when the pulp was instrumented after recording 80/80 with the electric pulp tester in symptomatic teeth. Cohen et al. (17), again, did not report on pain ratings experienced by patients during treatment. In our study, eight of nine of these patients received intrapulpal injections because of pulpal exposures. One patient received an intraosseous injection because the pulpal exposure was too small to adequately give an intrapulpal injection. Three of 8 (37.5%) patients

reported moderate pain when receiving the intrapulpal injection (Table 17). Dreven (29) reported that 6 of 30 (20%) patients reported moderate pain when receiving the periodontal ligament injection as a supplement to either the inferior alveolar injection or a maxillary infiltration in symptomatic teeth recording 80/80 with the electric pulp tester. Cohen et al. (17) reported on their impressions of the pain patients experienced when receiving a periodontal ligament injection and reported them as nonpainful. They did not report on the pain caused by intrapulpal injections. In our study, 5 maxillary and 3 mandibular posterior teeth required intrapulpal injections after recording negative responses to both the electric pulp tester and the Green Endo-Ice® after initial block injections. Dreven (29) reported that 2 maxillary and 4 mandibular teeth required supplemental periodontal ligament injections even after recording an 80/80 with the electric pulp tester due to pain during treatment. These results indicate that patients may experience moderate pain with an intrapulpal injection even after reporting clinical signs of a successful inferior alveolar nerve block, or recording an 80 with the electric pulp tester and testing negative to Green Endo-Ice®. This may not be acceptable, clinically, to most patients. In this study we decided to expedite treatment when the pulp was exposed by giving a supplemental intrapulpal injection rather than an intraosseous injection. This was done to simulate a clinical setting and to hasten treatment completion for the patients. This may not necessarily have been the best choice of treatment because of the moderate pain ratings recorded by the patients. Therefore, it is recommended that practitioners remove the rubber dam and administer an intraosseous injection instead of using an intrapulpal injection.

The pain ratings for the insertion of the needle for the infiltration injection, the deposition of anesthetic solution, the perforation of the cortical bone, and the deposition of the anesthetic solution for the Stabident intraosseous injection were evaluated using the numerical scale. The results are summarized in Tables 18-21.

Needle insertion into the attached gingiva at the site of perforation was rated moderate to severely painful by 3 of 24 (12.5%) patients (Table 18). This occurred even though an infiltration or inferior alveolar nerve block had been administered. Coggins (27) reported an overall pain rating of moderate to severe in 17% of patients when the Stabident injection was used as a primary injection. He reported that 8 of 40 (20%) mandibular first molars and 3 of 40 (8%) maxillary first molars gave a moderate pain rating. Replogle (28) reported a 2% (2 of 84) rating of moderate pain when the Stabident injection was used as a primary injection technique in asymptomatic mandibular first molars. Dunbar (26) reported 1 of 80 (1%) injections elicited a rating of moderate to severe pain on needle insertion when used after the inferior alveolar nerve block in mandibular first molars of asymptomatic patients. In this study a higher percentage of moderate to severe pain was recorded compared with Dunbar (26). Therefore, symptomatic patients may have a potential to experience more pain on needle insertion in mandibular molars. We see from Table 18 that 15% of mandibular molars and 0% of maxillary molars elicited moderate to severe pain ratings during the insertion of the needle at the perforation site. In comparing these results to the above three studies (26,27,28), differences in operator technique (avoiding contact with the periosteum), patient pool (symptomatic and anxious patients versus asymptomatic dental students), and whether the intraosseous injection was given as

a primary or supplemental injection may account for the differences. Therefore, in mandibular molars, there is a potential for moderate to severe pain on needle insertion approximately 12% of the time. Further research with a larger patient pool may also shed some light on the pain of needle insertion in symptomatic teeth. None of the studies utilized topical anesthetic in the area to be infiltrated, as recommended by the Stabident Manual (130). The use of topical anesthetic may or may not have any effect on the pain ratings in symptomatic patients. In asymptomatic teeth, Nist et al. (136), Yonchak (137), Mason (143), and Gill and Orr (179) have reported that topical anesthetic is ineffective clinically while Rosivack et al. (178) have reported on its effectiveness.

In comparing the pain ratings of mandibular and maxillary molars for needle insertion during the infiltration injection, Table 18 shows that the mandibular molars had 3 of 20 (15%) moderate to severe pain ratings versus 0 of 2 (0%) for maxillary molars. This difference is most likely due to the fact that the buccal attached gingiva of the maxillary molars achieves better anesthesia because of better diffusion of the anesthetic solution in the immediate area and due to numbing of the nerves supplying the area directly. The buccal gingiva of the mandibular molars may require a long buccal injection to gain anesthesia. Studies by McLean et al. (134), Vreeland et al. (133), and Hinkley et al. (135) have shown that some subjects achieved buccal anesthesia after only the inferior alveolar nerve block. It may be that the patients who experienced pain did not have long buccal anesthesia after the inferior alveolar nerve block. When comparing molars to premolars, one sees that 3 of 22 (14%) molars recorded moderate to severe pain and 0 of 2 (0%) premolars had moderate pain. This too relates to the innervation of the attached gingiva

and the type of anesthetic technique used. Mandibular premolar gingiva may gain some anesthesia due to numbing of the mental nerve when the inferior alveolar nerve is numbed.

The infiltration injection was given into attached gingiva at the site selected for cortical bone perforation. No attempt was made to avoid contacting periosteum while inserting the needle. Goaslind et al. (171) reported a mean thickness of 1.25 mm for attached gingiva, therefore avoiding contact with the periosteum during insertion of the needle would be very difficult. The Stabident Manual (130) offers an alternative site to infiltration in the attached gingiva. It states that infiltrating the alveolar mucosa and allowing 30 seconds for diffusion of the anesthetic solution will also help in reducing pain of perforation. This injection may be less painful since alveolar mucosal infiltrations avoid the periosteum (143,177) and have been reported as less painful than when periosteum is contacted (142,176). Another alternative would be the long buccal injection given for mandibular molars. It anesthetizes the buccal soft tissues of the molars (34) but may itself be painful.

The pain ratings for solution deposition during infiltration injections are summarized in Table 19. Two of 24 patients (8%) reported moderate to severe pain. Both of these were mandibular molars. Dunbar (26) reported a 5% rate of moderate to severe pain on solution deposition for the first molar following an inferior alveolar nerve block in asymptomatic subjects. Replogle (28) reported a 2% response of moderate to severe pain on infiltration (combined needle insertion and anesthetic deposition) in mandibular first molars when the Stabident injection was used as a primary injection. Coggins (27)

reported a 5% overall response of moderate to severe pain on solution deposition when the Stabident technique was used as a primary injection. He recorded 1 (3%) subject for the mandibular and 1 (3%) subject for the maxillary first molar sites. The results of these three studies (26,27,28) compare well with the results attained in this study utilizing symptomatic subjects. It can be concluded that the deposition of 0.1 ml of anesthetic solution results in no pain except for the mandibular molars where the potential for moderate to severe pain is 8%. Again, deposition of anesthetic solution may be less painful if an additional infiltration injection is given in the alveolar mucosa, as previously discussed and suggested by the Stabident Manual (130). A long buccal injection may also prove to be helpful.

Table 20 summarizes the pain ratings for perforation of the cortical bone using the Stabident perforator. Pain was rated moderate to severe in 2 of 24 (8%) of the perforations, both being in the maxillary arch, one for a molar and one for a premolar. Coggins (27) reported that, overall, 3% (4 of 160) of the perforations elicited moderate pain during perforation of the cortical bone. Two of these (50%) were in the mandibular first molar sites. Replogle (28) reported that 5-7% of the subjects reported moderate to severe pain in mandibular first molars. Both studies used the Stabident injection as a primary injection. Dunbar (26) reported an incidence of 0% moderate to severe pain on perforation following an inferior alveolar nerve block and buccal infiltration prior to perforation in the mandibular first molar region. All three of these studies utilized asymptomatic patients, while this study utilized symptomatic patients who were in pain, difficult to anesthetize, and probably anxious. This could explain the differences in the

percentages. Leonard (131) reported being able to perforate cortical bone without pain with the application of topical anesthetic for 50-60 seconds and no infiltration of the attached gingiva in most patients. However, he failed to report on how many patients did require buccal infiltrations due to perforation pain. It appears that, clinically, perforation of the cortical bone following an inferior alveolar nerve block in symptomatic patients is no more painful than perforation when the intraosseous injection is used as a primary injection or following an inferior alveolar nerve block and infiltration injection in asymptomatic patients.

The summary of pain ratings for solution deposition into the cancellous bone during the intraosseous injection are found in Table 21. One patient (4%) reported severe pain on solution deposition. This was a mandibular molar. Dunbar (26) reported that none of his subjects reported moderate to severe pain on solution deposition. Coggins (27) had a 15% incidence of moderate pain and Replogle (28) a 2-9% incidence of moderate to severe pain on solution deposition in the mandibular first molar region when the Stabident injection was used as a primary injection. It appears that anesthetic solution deposition for the Stabident injection, either as a primary or following an inferior alveolar nerve block in symptomatic or asymptomatic patients, may cause discomfort in some patients. If it does cause pain, it may be rated severe, but generally will be rated as none to mild.

The rate of deposition of solution has been implicated as a possible source of discomfort by the Stabident Manual (130). However, they did not state the ideal rate of solution deposition. It is unlikely that they would recommend a rate slower than used in this study (0.9 ml/minute). Replogle (28) used a rate of 1.8 ml over 2 minutes, Coggins

(27) a rate of 1.8 ml over 60 seconds, and Dunbar (26) a rate of 1.8 ml over 30 seconds. The rate of injection in this study was approximately 1.8 ml of anesthetic over 2 minutes. One can see from the previous paragraph that there was very little difference in the pain ratings for solution deposition during the Stabident injection between the four studies. It appears that the rate of deposition of anesthetic solution has little effect on the pain ratings recorded.

The presence of back-pressure during the intraosseous injection was recorded in this study. Back-pressure was defined as the need for greater than light finger pressure to inject into the cancellous bone. Back-pressure was encountered in 3 of 24 (12.5%) injections (Table 22). Two of these sites were in the mandible. Inadequate perforation of the cortical bone, perforation into or against the periodontal ligament or lamina dura, or a clogged needle are possible causes for back-pressure. In all 3 cases, reperforation of the original site alleviated the problem. Apparently the reperforation gained entrance to the cancellous bone. The Stabident Manual (130) describes the molar regions as the most difficult areas to perforate due to access and the thickness of cortical bone, especially in the mandibular molar region. Replogle (28) reported a 19% incidence of back-pressure. Coggins (27) reported a 9% overall rate of back-pressure with 50% of these occurring in the mandibular first molar area and 21% in the maxillary first molar area. Dunbar (27) reported a 15% rate. It should be noted that all 3 of these studies limited their perforations to the distal of mandibular or maxillary first molars, and these areas, as previously stated, may be the most difficult areas to perforate. In this study, if a second or third molar was used, perforation was made mesial to the tooth due to difficulties with

access. This would not have an effect on the back-pressure results for the second molars since this area is the same location as a perforation for a first molar. However, there may be a chance of an increase in the number of cases exhibiting back-pressure for perforation sites distal to the first/second molar interproximal site due to an increase in thickness of the cortical bone (169). The effect of using a site mesial to the test tooth on the efficacy of the Stabident injection has not been reported. However, we assumed that there would be no difference in the second or third molars because of the results of Dunbar (26), Coggins (27) and Replogle (28) who reported that the tooth immediately distal to the perforation/injection site gained anesthesia at the same rate as the test tooth.

An increase in heart rate as reported by the patients, was assessed subjectively in this study and is summarized in Table 23. Subjects were questioned during and for 2 minutes after the intraosseous injection if they felt any heart rate increases. Patients reported an increase during or after 11 of 24 (46%) intraosseous injections. Nine of the responses occurred in the mandible and 2 in the maxilla. Replogle (28) reported that approximately 67% of her subjects had a measurable increase in heart rate during or 2 minutes after receiving a Stabident injection of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine. However, only 57% of the subjects reported the feeling of a subjective rate increase. No report of heart rate increase was found with 3% mepivacaine. Coggins (27) and Dunbar (26) reported that approximately 75-78% of their subjects reported a subjective increase in heart rate. The lower percentage of positive responses in this study may be due to the subject pool and the number of subjects studied. The above three studies (26,27,28) used many more teeth and asymptomatic dental students; while this study utilized a limited

number of symptomatic emergency patients. Anxiety about treatment and the fact that the patients were already in pain may have effected the subjective heart rate response of the Stabident injection.

The intraosseous injection, as reported by Smith and Pashley (117), has a cardiovascular effect and it would be expected that an increased heart rate would occur no matter where the injection site is as long as the anesthetic solution is deposited intraosseously. Lilienthal (56,126), Lilienthal and Reynolds (55), Canell and Cannon (57), and Pearce (128) all reported mild increases in heart rate in patients when using the intraosseous injection with anesthetic solutions containing a vasoconstrictor. Replogle (28), Lilienthal (56,126) and Lilienthal and Reynolds (55) reported that the heart rates of the patients generally returned to baseline values within 2-3 minutes following the intraosseous injection of a vasoconstrictor containing anesthetic. Lilienthal (56) reported an increase in diastolic blood pressure and heart rate within 10-30 seconds of delivering an intraosseous injection of as little as 0.9 ml of 4% prilocaine with 1:200,000 epinephrine. This may indicate that it may not be the concentration or volume of epinephrine that causes an increase in heart rate, but merely the presence of epinephrine in the cancellous space. Replogle (28) did report, however, that approximately 14% of her subjects had a mean rise in heart rate of 37 beats above baseline level coupled with a prolonged effect of up to 22 minutes. She classified these subjects as possibly "epinephrine sensitive" in that the beta effects of the epinephrine caused a higher cardiac rate and/or prolonged effect as compared to other subjects.

The increased heart rate has been attributed to the introduction of exogenous epinephrine from the anesthetic solution (55,56,57). The present study only reported the occurrence of a subjective increase in heart rate. It did not measure the rate nor duration of the heart rate. Clinically, it appears that, in healthy symptomatic patients, the intraosseous injection of 1.8 ml of 2% lidocaine with 1:100,000 epinephrine over a 2 minute time period is safe. However, patients should be warned of this potential increase in heart rate to reduce their anxiety.

Table 24 summarizes the results of testing, postintraosseous injection, with both the Analytic Technology® electric pulp tester and the Green Endo-Ice®. At minute 1, 92% (22 of 24) of the teeth tested 80/80 with the electric pulp tester and negative to the Green Endo-Ice®. By minute 2, all the patients failed to respond to the Green Endo-Ice® and by the end of the testing period, 96% of the teeth recorded 80/80 readings. Coggins (27) and Replogle (28) both reported an immediate rate of onset when using the Stabident injection as a primary injection. Dunbar (26) reported that the onset of pulpal anesthesia in mandibular first molars was immediate when the Stabident injection was added to the inferior alveolar nerve block. He postulated that after the completion of the intraosseous injection, treatment could begin without a waiting period. Lilienthal (126), Magnes (125), Pearce (128) and Bourke (123) also suggested that treatment may begin immediately after the intraosseous injection is given with the assurance of profound pulpal anesthesia. Leonard (131) began extraction procedures on teeth primarily anesthetized with the Stabident injection within 15 seconds of completion of the injection. The results recorded in this study using the electric pulp tester and Green Endo-Ice® also indicate that pulpal

anesthesia is essentially immediate (within 1-3 minutes) in symptomatic patients, when anesthesia is achieved.

After receiving the Stabident intraosseous injection and being tested for 3 minutes, in all 24 cases endodontic treatment was started. One patient (mandibular first molar) did not attain an 80/80 reading with the electric pulp tester after minute 3 but did test negative to the Green Endo-Ice® (Table 24,25). It was decided to continue with endodontic treatment to see which of the two tests would be correct in determining pulpal anesthesia. The patient did have pain on further treatment and did require further supplemental anesthesia. In this case the electric pulp tester was shown to be more accurate than the Green Endo-Ice® in determining pulpal anesthesia. This follows the results seen after the initial anesthesia injections where 42% of mandibular teeth recorded an 80, but 81% were negative to Green Endo-Ice® and it may confirm that the electric pulp tester is more accurate in determining pulpal anesthesia than Green Endo-Ice®.

Table 25 shows that 1 of 24 (4%) patients receiving an intraosseous injection failed to test negative to both the electric pulp tester and Green Endo-Ice®. This patient tested positive to the electric pulp tester but negative to the Green Endo-Ice® and treatment was continued as described in the previous paragraph.

Six of the 24 (25%) patients receiving the Stabident intraosseous injection reported some discomfort during endodontic treatment (Table 26). Four of 23 (17%) patients had pain when the pulp was exposed (all mandibular molars), 1 of 23 (4%), a maxillary molar, when the pulp tissue was removed and files were placed to length (Table 28). One patient reported pain when dentin was entered (Table 27). Two of the patients received a second

intraosseous injection while the remaining 4 received intrapulpal injections due to the pulp being exposed (Table 29). These results indicate that the electric pulp tester was 79% (19 of 24) effective in determining pulpal anesthesia versus Green Endo-Ice® which was 75% (18 of 24) accurate. It can be concluded that both tests were equal in measuring pulpal anesthesia after teeth received Stabident intraosseous injections. However, neither test was one hundred percent accurate in ensuring pulpal anesthesia.

The definition for success of the intraosseous injection was that the patient experienced no pain during endodontic therapy after receiving the Stabident injection. However, it was discovered that one patient, who reported mild pain when the pulp was exposed, reported no pain on receiving an intrapulpal injection. It has been the clinical experience of this operator that patients will sometimes respond to the initial exposure of the pulp, but have no further pain experiences during treatment. This phenomenon has not been reported and some possible explanations come to mind. First and foremost is that during inflammation of the pulp, blood vessels dilate and vascular permeability increases due to the release of histamine, bradykinin, serotonin, prostaglandins and leukotrienes (15). This, in turn, leads to elevated capillary pressure and, combined with increased vascular permeability, then leads to increased fluid filtration into the surrounding tissue (edema). Increased tissue pressure and the fact that the pulp is encased in a noncompliant environment leads to the spread of inflammation throughout the pulp (15). Perhaps when the roof of the pulp chamber is perforated, the quick release of this pressure is felt by the patient as the fluid and blood find release into the access cavity and the pain fibers (Aδ-, C-fibers) respond. The second explanation is that the patient is responding to the breakthrough of the roof of

the pulp chamber. As the operator applies pressure to the handpiece during access of a vital case the sudden release or breakthrough may startle the patient and be perceived and reported as pain, although, clinically it seems that the patient does experience pain. Again these are only speculations and future studies may be conducted to determine why this phenomenon occurs. In our study, this patient was considered a success for the Stabident injection since the pain was mild upon access and no pain was elicited upon the intrapulpal injection.

A second patient experienced moderate pain during pulp tissue removal 11 minutes and 30 seconds after the intraosseous injection was given (Table 33). Dunbar (27) reported a 10% occurrence of short duration of anesthesia (less than 60 minutes) in mandibular teeth using an inferior alveolar nerve block and intraosseous injection. Coggins (26) reported that 60% of maxillary and mandibular first molars, and Replogle (28) that 77% of mandibular first molars, had lost anesthesia after 1 hour when the intraosseous injection was given as a primary injection. At the 10-12 minute mark, Replogle (28) had approximately 60% and Coggins (27) 75% of mandibular molars recording 80 readings with the electric pulp tester. All of these studies utilized asymptomatic teeth. In this study vital, symptomatic teeth were used and the effects of the pulpal inflammation on the duration of pulpal anesthesia is not known. This patient was most likely beginning to lose anesthesia since she was numb initially when the pulp chamber was accessed and files placed to length. At this point, the operator needs to decide whether to give an intrapulpal injection or remove the rubber dam and give a second intraosseous injection. The intrapulpal injection is the more convenient injection to give, but will generally cause

moderate pain. This patient reported no pain on receiving the intrapulpal injection. This could be due to the patient entering a phase of anesthesia when previously she may have lost it due to noncontinuous anesthesia. The patient did not experience any further pain during treatment and may have achieved anesthesia with the intrapulpal injection. The patient was considered a success due to the fact that the intrapulpal injection was rated as painless.

The pain ratings reported by patients during endodontic therapy after receiving the intraosseous injection are summarized in Table 30. Four of 6 (67%) patients had moderate pain during endodontic treatment. None of the patients reported severe pain. Previously in this study a rate of 73% moderate to severe pain was reported by patients during endodontic therapy after receiving only the mandibular nerve block or maxillary infiltration injection. None of the intrapulpal injections elicited a moderate or severe pain response after the intraosseous injection as compared to the 37.5% reported after the initial anesthesia injections. It may be concluded from these results that the intraosseous injection may reduce the pain of an intrapulpal injection.

Two patients received second intraosseous injections (Table 29). In one case, a mandibular first molar, a new perforation was made mesial to the tooth instead of reperforation of the distal cortical bone. In the second case, a mandibular second molar, a mesial perforation was made more apical than the first perforation. All second and third molars received the intraosseous injections mesial to the tooth because of access difficulties and the fact that the distal sites may have had cortical bone too thick to perforate (169). The Stabident Manual (130) reports that access can be a factor in

perforation site selection. The second case was successful in gaining pulpal anesthesia (Tables 31 and 32). In the first case, the patient never recorded an 80/80 with the electric pulp tester and had pain on exposure of the pulp and required an intrapulpal injection which was rated as moderately painful (Tables 31-33). This patient was considered a failure. The location of a second injection can only be speculated on. If the operator feels that the initial perforation did not pass through the cortical plate and allow anesthetic solution to enter the cancellous bone, a second perforation apical to the initial site may be successful. If, however, the operator felt that a good perforation was achieved, reperforation and injection may lead to the anesthetic leaking out of the first perforation. In this case, perforation of the opposing site may be indicated if its accessible.

Table 33 reviews the failures of the Stabident injections. Patients 7 and 36 both experienced pain with endodontic treatment. Patient 7 had moderate pain when the pulp was removed from a maxillary molar. Patient 36 had mild pain upon entering the pulp chamber of a mandibular molar. Both experienced mild pain when receiving an intrapulpal injection (Table 33). Patient 8 was the final failure (described in the above paragraph). This patient required a second intraosseous injection which was unsuccessful in gaining a negative response with the electric pulp tester and the patient experienced moderate pain on entering the pulp (Tables 31 and 32). This patient also experienced moderate pain on receiving the intrapulpal injection (Table 33). Cohen et al. (17) reported that 1 of 6 (17%) patients still felt pain during endodontic therapy even after testing negative to DDM following a second series of periodontal ligament injections. Emergency treatment in our study took approximately 30-45 minutes, on average, depending on the tooth type and

difficulty. Dunbar (26) reported that 90% of subjects were still numb after 60 minutes in asymptomatic patients receiving an intraosseous injection after the inferior alveolar nerve block. None of the patients that were considered failures had treatment that went beyond 60 minutes. Possible causes for failure of the intraosseous injection include: failure to gain a good perforation and injection of anesthetic solution into the cancellous space; anesthetic solution leaking back through the perforation; anatomic variances such as constricted cancellous spaces which prevent the anesthetic from reaching the apex; and apical inflammatory changes that prevent the anesthetic solution from being effective.

Table 34 summarizes the success of the Stabident intraosseous injection system. Twenty-one of 24 (87.5%) intraosseous injections were successful in gaining pulpal anesthesia for endodontic therapy in posterior teeth diagnosed with irreversible pulpitis. The rate of success for mandibular teeth was 90.5% and 67% for maxillary teeth. A statistical comparison of the arches (Fisher Exact Test = 0.343) revealed no significant differences in success between the two arches. The Fisher Exact Test was run because the sample size for the maxillary teeth was very small and due to this, the power of the value may be low. Coggins (27) and Replogle (28) reported success rates of : 90% in maxillary lateral incisors; 93% in maxillary first molars; 78% in mandibular lateral incisors; and 75% in mandibular first molars utilizing asymptomatic teeth with the intraosseous injection being the primary injection. Replogle (28) also reported a success rate of 45% using 3% mepivacaine for mandibular first molars. Dunbar (26) reported a 90% success rate (numb within 15 minutes and numb for 1 hour) in asymptomatic mandibular first molars when using the Stabident injection as a supplement to the inferior alveolar nerve block. Leonard

(131) reported a success rate of 87.5% (56 of 64 having excellent anesthesia) for extraction of teeth using the Stabident injection as the primary source of anesthesia. Cohen et al. (17) reported an 96% success rate using the periodontal ligament injection as a supplement for mandibular first molars diagnosed with irreversible pulpitis. However, in their study they performed only a pulpotomy which may have been a shorter time span for treatment. They also delivered 4 periodontal ligament injections each time the supplemental injection was indicated. Walton and Abbott (20) reported a 92% success rate using the periodontal ligament injection as a supplemental technique for teeth requiring restorative or endodontic treatment. They did not separate the two categories. All of these studies utilized 2% lidocaine with 1:100,000 epinephrine. Lilienthal (126) utilized 2% lidocaine with 1:80,000 noradrenalin to achieve immediate and profound anesthesia of maxillary and mandibular teeth with the intraosseous injection. Magnes (125) reported a 95-99% success rate when using 2% lidocaine with 1:100,000 epinephrine in 1,800 patients on mandibular anterior teeth. Pearce (128), in a clinical study, reported a success rate of 90% when using the intraosseous injection as a supplement to the mandibular nerve block in patients undergoing endodontic treatment. The results of this study support the results of these other studies. It appears that the Stabident intraosseous injection is as effective as the periodontal ligament injection in gaining pulpal anesthesia in both symptomatic and asymptomatic teeth. Additionally, the intraosseous injection may be less painful (Tables 18-21) when compared to the pain ratings reported for the periodontal ligament injection (11,21,24). The Stabident intraosseous injection is also able to deliver more anesthetic solution into the cancellous

space as compared to the periodontal ligament injection. This could mean that there is a greater chance for anesthetic to reach the apex of the target tooth. The duration of the Stabident intraosseous injection is also longer (27) than the 4-10 minute duration reported for the periodontal ligament injection (21,24). It therefore can be used as a primary injection for short duration treatments. One of the drawbacks of the intraosseous injection includes the fact that the rubber dam needs to be removed to give the injection. The periodontal ligament injection may be delivered with the rubber dam in place which may expedite treatment. Another disadvantage is the cardiac effects that occur when using epinephrine containing anesthetic solution in the intraosseous injection. Whether solutions containing other vasoconstrictors would be useful in the intraosseous injection must await further research. The use of the Stabident intraosseous injection utilizing epinephrine containing anesthetics is contraindicated in patients with significant cardiac disease, angina, or arrhythmia's. Also, patients on MAO inhibitors, tricyclic antidepressants, beta-blockers, and phenothiazines should receive the intraosseous injection with caution. Finally, the Stabident intraosseous injection has certain postoperative problems unique to the injection. These include postoperative tooth tenderness, pain or swelling of the gingiva at the injection site, and formation of a draining fistula at the injection site. However, these complications usually resolve or are successfully treated with antibiotics.

Other types of supplemental injection have been given in the hopes of improving the anesthetic effectiveness of the standard inferior alveolar nerve block. Nist et al. (136) compared the success of a mandibular nerve block alone to a mandibular nerve block with an incisive nerve block in human mandibular teeth. They reported a 70% success in the

mandibular first molar using the combination of injections. Childers (122) added the periodontal ligament injection to the inferior alveolar nerve block and increased the rate of success in first molars from 62.5% (mandibular nerve block alone) to 77.5%. Our study reveals that adding the Stabident intraosseous injection to the inferior alveolar nerve block increased the success in gaining pulpal anesthesia in symptomatic patients from 64% to 87.5%. We concluded from our results, and those of Dunbar (26), that the use of the intraosseous injection would result in a greater percentage of successful pulpal anesthesia compared to the periodontal ligament injection and incisive nerve block in mandibular posterior teeth when used as supplemental injection to the inferior alveolar nerve block. The use of the other injections may not be required if a successful intraosseous injection is given. The Stabident injection may be used in partially necrotic teeth (teeth with a necrotic chamber but vital apical canals) since the difficulty in gaining good anesthesia in these cases also exists. Its use in symptomatic necrotic teeth needs to be studied. Injection of the anesthetic solution into the cancellous space may not achieve the same level of anesthesia and may be more painful in symptomatic necrotic teeth. This is due to an increase in vascularity of the area which would lead to faster removal of the anesthetic solution and injection of a solution into the area would increase tissue pressure and cause more pain.

In this study, no post-treatment questionnaires or follow-ups were made. The patients were advised to return to the Graduate Endodontic clinic if any postoperative problems occurred. The patients were also given appropriate pain medications. None of the 24 patients receiving the intraosseous injection called or returned for further emergency

treatment as related to the intraosseous injection. Replogle (28), Dunbar (26) and Coggins (27) reported a 3-10% rate of moderate postoperative pain when the Stabident injection was used as either a primary or supplemental injection in asymptomatic patients. Approximately 4% of their patients developed an exudative or localized swelling at the site of perforation. All of these cases resolved within 1-4 weeks or after the patient was placed on antibiotics. The investigators postulated that the damage was due to overheating of the bone caused by excessive pressure during perforation. Bourke (123) has reported on the effects of the intraosseous injection on the gingiva, periosteum, cortical bone or cancellous bone. He has reported on his clinical findings of over 20 years of using the intraosseous injection and states that the tissue damage caused by the intraosseous injection is mild and reversible. He also reported that the damage the gingiva and bone incur is the result of excess speed of rotation of the perforator and excessive pressure on the handpiece which causes necrosis due to overheating. The Stabident Manual (130) suggests the use of a surface disinfectant prior to the injection. This may lower or prevent the incidence of infection of the gingival perforation, but it will not prevent the necrosis of bone or soft tissue due to overheating of these tissues. Further study is needed on the histological effects of the Stabident intraosseous injection. In this study, patients may have experienced postoperative pain due to the intraosseous injection but it was masked by postoperative endodontic pain or identified as postendodontic treatment pain.

Dunbar (26), Coggins (27) and Replogle (28) have reported on some of the symptoms associated postoperatively with the Stabident intraosseous injection. They reported a 4-

11% rate (27,28) of subjects feeling that the test tooth felt high in occlusion the day following the injection. This feeling was reported to last 3-7 days. White et al. (21) and Schleder et al. (24) reported, when using the periodontal ligament injection, that 36% and 49.3% of patients, respectively, reported feeling that their tooth was high in occlusion. Replogle (28) also reported that 8% of patients had feelings of gum sensitivity to tooth brushing or even localized edema of the gingival tissue at the site of perforation. Coggins (27) reported having subjects with swollen and puffy papilla. Patients may have postoperative swellings and exudate at the site of the intraosseous perforation approximately 4% the time (26,27,28). There may also be signs of purulent drainage or the formation of a fistula at the perforation site (27,28). Treatment of these postoperative problems includes placing the patients on an antibiotic regimen and having them use a Chlorhexidine rinse. The problems have been reported to resolve within 1-4 weeks and, in all the cases, no detectable defects were noted. The practitioner should be aware of these possible postoperative problems and advise the patient that they may occur. The patient should be informed to return to the clinic for follow-up evaluation if the signs or symptoms present themselves.

CHAPTER VI

SUMMARY AND CONCLUSIONS

The purpose of this study was to evaluate the anesthetic effectiveness of the Stabident intraosseous injection, used as a supplemental technique, in achieving pulpal anesthesia in teeth diagnosed as having irreversible pulpitis. The teeth studied were maxillary and mandibular posterior teeth. Additionally, this study evaluated the use of Green Endo-Ice® and the Analytic Technology® electric pulp tester in determining pulpal anesthesia.

The Stabident intraosseous injection was found to be 90.5% effective in mandibular posterior teeth and 67% effective in maxillary posterior teeth. The overall success rate was 87.5%.

Green Endo-Ice® was found to be in moderate agreement (80.39%, kappa value = 0.617, phi coefficient = 0.648) overall with the electric pulp tester in determining pulpal anesthesia. However, in mandibular posterior teeth, the electric pulp tester was found to be more reliable than the Green Endo-Ice® in determining pulpal anesthesia. Neither testing modality was found to be 100% reliable.

Our study found that significantly more mandibular teeth, diagnosed with irreversible pulpitis, required intraosseous supplemental anesthesia as compared to maxillary teeth ($p = 0.0000$). This result confirms previous reports that mandibular anesthesia in symptomatic

teeth is difficult to attain with an inferior alveolar nerve block alone. Our study also supports previous findings that even with subjective signs of anesthesia, an 80/80 response to the electric pulp tester and no response to Green Endo-Ice®, complete pulpal anesthesia is not guaranteed in symptomatic posterior teeth. The use of the Stabident intraosseous injection reduced the number of failures from 46% with inferior alveolar nerve block alone to 9.5% when the intraosseous injection was added as a supplemental technique.

The pain ratings for the intraosseous injection in symptomatic patients compared favorably with the pain ratings reported for asymptomatic patients receiving the intraosseous injection. Generally, they were considered mildly painful. It was also found that a successful intraosseous injection reduced the pain rating of intrapulpal injections from moderate to severe to either mild or no pain.

In conclusion, the Stabident intraosseous injection system is a valuable supplemental anesthetic technique to the inferior alveolar nerve block and maxillary infiltration for posterior teeth diagnosed with irreversible pulpitis.

Table 1 Biographical Data for All Subjects

	Mean Age (years)	Male No.	Male %	Female No.	Female %
IAN/Inf.	35.3 (19-68)	13	48%	14	52%
IAN/Inf. + IOI	32.6 (19-63)	10	42%	14	58%
Total	34.0 (19-68)	23	45%	28	55%

Table 2 Distribution of Teeth for All Subjects

Arch	Premolars		Molars			Total
	First	Second	First	Second	Third	
Mandibular	1	1	12	11	1	26
Maxillary	1	10	12	2	0	25
Total	2	11	24	13	1	51
Percent	25%		75%			100%

Table 3 Clinical Diagnostic Data for All Subjects

	Preoperative Pain Ratings of Test Tooth			Mean Baseline EPT Reading	EPT Pain Ratings*				Endo-Ice Results		Endo-Ice Pain Ratings*			
	mild (1)	moderate (2)	severe (3)		none (0)	mild (1)	moderate (2)	severe (3)	(+)	(-)	none (0)	mild (1)	moderate (2)	severe (3)
Total	8	19	24	39.5	0	10	2	2	49	2	1	0	2	11
	(+) = 51; (-) = 0													
Mean	2.3				1.4						2.6			
Median	2				1						3			

* Data collected starting with patient #38. N=14

Pain Ratings EPT vs. Endo-Ice, $p = 0.0039$, Test Statistic = -31, median difference = -2, intraquartile = 2.

Table 4 Summary of Pain Ratings for Needle Insertion During IANB/Infiltration Injections Utilizing the Numerical Scale

Injection Type	None	Mild	Moderate	Severe
Inferior Alveolar Nerve Block	5 (19%)	15 (58%)	6 (23%)	0 (0%)
Infiltration	11 (44%)	12 (48%)	2 (8%)	0 (0%)

Table 5 Summary of Pain Ratings for Placement of Needle During IANB/Inf. Injections Utilizing the Numerical Scale

Injection Type	None	Mild	Moderate	Severe
Inferior Alveolar Nerve Block	8 (31%)	9 (35%)	5 (19%)	4 (15%)
Infiltration	15 (60%)	10 (40%)	0 (0%)	0 (0%)

Table 6 Summary of Pain Ratings for Solution Deposition During IANB/Inf. Injections Utilizing the Numerical Scale

Injection Type	None	Mild	Moderate	Severe
Inferior Alveolar Nerve Block	11 (42%)	7 (27%)	6 (23%)	2 (8%)
Infiltration	17 (68%)	6 (24%)	2 (8%)	0 (0%)

Table 7 Time and Pulpal Anesthesia for All Experimental Teeth

Postinjection Time	Mandibular*		Maxillary**	
	EPT	Endo-Ice	EPT	Endo-Ice
1	5 (19%)	11 (42%)	15 (60%)	21 (84%)
2	9 (35%)	14 (54%)	21 (84%)	24 (96%)
3	9 (35%)	18 (69%)	23 (92%)	23 (92%)
4	11 (42%)	21 (81%)		
5	12 (46%)	21 (81%)		
Combined Total After Testing Periods			EPT - 35/51 (67%) Endo-Ice - 44/51 (86%)	

* N=26
** N=25

Table 8 Comparison of Responses of Electric Pulp Tester Versus Green Endo-Ice

	No. of Negative Responses with Green Endo-Ice	No. of Positive Responses with Green Endo-Ice
When No Response with EPT:	22	1
When Response with EPT:	9	19

chi square = 21.367, df = 1, p = 0.0001
phi coefficient = 0.648

Percent agreement = 80.39%
Kappa value = 0.617

Table 9 Summary of Pain Ratings for EPT/Endo-Ice Testing after IANB/Infiltration Injections Utilizing the Numerical Scale*

Time	EPT None (80/80)	EPT Mild	EPT Moderate	EPT Severe	Endo-Ice None (no response)	Endo-Ice Mild	Endo-Ice Moderate	Endo-Ice Severe
1**	8 (53%)	6 (40%)	1 (7%)	0 (0%)	12 (80%)	0 (0%)	1 (7%)	2 (13%)
2**	11 (73%)	4 (27%)	0 (0%)	0 (0%)	13 (87%)	1 (7%)	0 (0%)	1 (7%)
3**	12 (80%)	3 (20%)	0 (0%)	0 (0%)	13 (87%)	2 (13%)	0 (0%)	0 (0%)
4***	4 (67%)	2 (33%)	0 (0%)	0 (0%)	4 (67%)	2 (33%)	0 (0%)	0 (0%)
5***	5 (83%)	1 (17%)	0 (0%)	0 (0%)	4 (67%)	2 (33%)	0 (0%)	0 (0%)

* Data recording began with patient #37. N=15
** Combined maxillary and mandibular data
*** Mandibular teeth only. N=7

Table 10 Summary of Pulp Test Results in Teeth Positive to EPT, Green Endo-Ice, or both after IANB/Infiltration Injections

Arch	Total No. of Pt.s and Precentage	Positive Response To:				No. Requiring Intraosseous Injection Prior to Access
		EPT alone	Endo-Ice alone	Both Tests	Total	
Maxilla	25 (49%)	0 (0%)	0 (0%)	2 (8%)	2 (8%)	2/25 (8%)
Mandible	26 (51%)	11 (42%)	2 (7%)	3 (11%)	16 (61%)	16/26 (61%)
Total	51 (100%)	11 (22%)	2 (4%)	5 (10%)	18 (35%)	18/51 (35%)

Table 11 Summary of Clinical Results in Teeth Negative to EPT and Green Endo-Ice after IANB/Inf. Injections

Arch	Total No. of Teeth	No. of Teeth Negative to both EPT/Endo-Ice	No. of Teeth Requiring Supplemental Anesthesia
Maxilla	25	23/25 (92%)	6/23 (26%)
Mandible	26	10/26 (38%)	8/10 (80%)
Total	51	33/51 (65%)	14/33 (42%)

Table 12 Summary of Clinical Results during Endodontic Access in Teeth Negative to EPT/Green Endo-Ice after IANB/Inf. Injections

Arch	No. of Teeth Negative to both EPT/Endo-Ice	Pain upon Entering Dentin	No. Requiring Intraosseous Injection due to pain in dentin
Maxilla	23	1/23 (4%)	1 (4%)
Mandible	10	4/10 (40%)	4 (40%)
Total	33	5/33 (15%)	5 (15%)

Table 13 Summary of Clinical Results upon Pulp Exposure, Pulp Removal, or Filing in Teeth Negative to EPT/Green Endo-Ice after IANB/Inf. Injection

Arch	No. of Teeth Negative to both EPT/Endo-Ice	Pain upon Pulp Exposure	Pain upon Removing Pulp	Pain on Filing	Total No. with Pain	No. Requiring Intrapulpal Inj. due to pulpal pain	No. Requiring Intraosseous Inj. due to small exposure
Maxilla	23	1 (4%)	3 (13%)	1 (4%)	5 (22%)	5 (22%)	0 (0%)
Mandible	10	2 (20%)	2 (20%)	0 (0%)	4 (40%)	3 (30%)	1 (10%)
Total	33	3 (9%)	5 (15%)	1 (3%)	9 (27%)	8 (24%)	1 (3%)

Table 14 Summary of Teeth Requiring Supplemental Anesthesia after IANB/Inf. Injection

Arch	Total No. of Teeth	No. Requiring Intraosseous Inj. prior to access	No. Requiring Intraosseous Inj. during tx.	Total No. Requiring Intraosseous Inj.	Total No. Requiring Intrapulpal Inj.	Total No. Requiring Intaosseous and/or Intrapulpal Inj.
Maxilla	25	2/25 (8%)	1/25 (4%)	3/25 (12%)	5/25 (20%)	8/25 (32%)
Mandible	26	16/26 (61%)	5/26 (19%)	21/26 (81%)	3/26 (12%)	24/26 (92%)
Total	51	18/51 (35%)	6/51 (12%)	24/51 (47%)	8/51 (16%)	32/51 (63%)

Table 15 Distribution of Teeth Requiring Intraosseous Injection

Arch	Premolars		Molars			Total
	First	Second	First	Second	Third	
Mandibular	1	0	9	10	1	21/26 (80.77%)*
Maxillary	0	1	1	1	0	3/25 (12.00%)*
Total	1	1	10	11	1	24/51 (47.05%)

* mandibular vs. maxillary arch: chi-square = 24.193, df = 1, p = 0.0000

Table 16 Pain Ratings for Subjects Who Felt Pain During Initial Access/Endodontic Treatment Prior to Supplemental Anesthesia

Location	Number	Mild	Moderate	Severe
Pain entering dentin	5 (36%)	1 (7%)	4 (29%)	0 (0%)
Pain entering pulp	4 (29%)	1 (7%)	1 (7%)	1 (7%)
Pain removing pulp	4 (29%)	1 (7%)	3 (21%)	1 (7%)
Pain on filing	1 (7%)	1 (7%)	0 (0%)	0 (0%)
Total	14 (100%)	4 (29%)	8 (57%)	2 (14%)

N=15

Table 17 Summary of Pain Ratings of Intrapulpal Injections After IANB/Infiltration Injections (No IOI Injections) Utilizing the Numerical Scale

	None	Mild	Moderate	Severe
Maxillary	1 (12.5%)	3 (37.5%)	1 (12.5%)	0 (0%)
Mandibular	0 (0%)	1 (12.5%)	2 (25%)	0 (0%)
Total	1 (12.5%)	4 (50%)	3 (37.5%)	0 (0%)

N=8

Table 18 Summary of Pain Ratings for Needle Insertion During Infiltration Injections Utilizing the Numerical Scale

Tooth	Number	None	Mild	Moderate	Severe
Mandibular Molars	20	8 (40%)	9 (45%)	2 (10%)	1 (5%)
Maxillary Molars	2	1 (50%)	1 (50%)	0 (0%)	0 (0%)
Mandibular Premolars	1	0 (0%)	1 (100%)	0 (0%)	0 (0%)
Maxillary Premolars	1	1 (100%)	0 (0%)	0 (0%)	0 (0%)
Total	24	10 (42%)	11 (46%)	2 (8%)	1 (4%)

Table 19 Summary of Pain Rating for Solution Deposition During Infiltration Injections Utilizing the Numerical Scale

Tooth	Number	None	Mild	Moderate	Severe
Mandibular Molars	20	18 (90%)	0 (0%)	1 (5%)	1 (5%)
Maxillary Molars	2	2 (100%)	0 (0%)	0 (0%)	0 (0%)
Mandibular Premolars	1	1 (100%)	0 (0%)	0 (0%)	0 (0%)
Maxillary Premolars	1	1 (100%)	0 (0%)	0 (0%)	0 (0%)
Total	24	22 (92%)	0 (0%)	1 (4%)	1 (4%)

Table 20 Summary of Pain Ratings for Perforation During Intraosseous Injections Utilizing the Numerical Scale

Tooth	Number	None	Mild	Moderate	Severe
Mandibular Molars	20	16 (80%)	4 (20%)	0 (0%)	0 (0%)
Maxillary Molars	2	0 (0%)	1 (50%)	0 (0%)	1 (50%)
Mandibular Premolars	1	1 (100%)	0 (0%)	0 (0%)	0 (0%)
Maxillary Premolars	1	0 (0%)	0 (0%)	1 (100%)	0 (0%)
Total	24	17 (71%)	5 (21%)	1 (4%)	1 (4%)

Table 21 Summary of Pain Ratings for Solution Deposition of Intraosseous Injections Utilizing the Numerical Scale

Tooth	Number	None	Mild	Moderate	Severe
Mandibular Molars	20	15 (75%)	4 (20%)	0 (0%)	1 (5%)
Maxillary Molars	2	1 (50%)	1 (50%)	0 (0%)	0 (0%)
Mandibular Premolars	1	1 (100%)	0 (0%)	0 (0%)	0 (0%)
Maxillary Premolars	1	1 (100%)	0 (0%)	0 (0%)	0 (0%)
Total	24	18 (75%)	5 (21%)	0 (0%)	1 (4%)

Table 22 Summary of Back-Pressure During the Intraosseous Injections

Arch	Back-Pressure on Injection
Maxilla	1 (33%)
Mandible	2 (10%)
Total	3/24 (12.5%)

Maxilla, N=3
Mandible, N=21

Table 23 Summary of an Increased Heart Rate During the Intraosseous Injections

Arch	Subjective Heart Rate Increase
Maxilla	2/3 (67%)
Mandible	9/21 (43%)
Total	11/24 (46%)

Maxilla, N=3
Mandible, N=21

Table 24 Time and Pulpal Anesthesia for All Experimental Teeth Receiving Intraosseous Injections

Postinjection Time	EPT	Endo-Ice
1	22 (92%)	22 (92%)
2	22 (92%)	24 (100%)
3	23 (96%)	24 (100%)

N=24

Table 25

Summary of Pulp Test Results in Teeth Positive to EPT, Green Endo-Ice, or both after Initial Intraosseous Injection

Arch	Total No. of Pt.'s and Percentages	Positive Response To:				No. Requiring Second Intraosseous Inj. Prior to Access
		EPT alone	Endo-Ice alone	Both Tests	Total	
Maxilla	3 (12.5%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Mandible	21 (87.5%)	1 (5%)	0 (0%)	0 (0%)	1 (5%)	0 (0%)*
Total	24 (100%)	1 (4%)	0 (0%)	0 (0%)	1 (4%)	0 (0%)*

* Patient received treatment despite result of pulp test.

Table 26 Summary of Clinical Results in Teeth Negative to EPT and Green Endo-Ice after Supplemental IOI Injection

Arch	Total No. of Teeth	No. of Teeth Negative to both EPT/Endo-Ice	No. of Teeth Requiring Supplemental Anesthesia
Maxilla	3	3/3 (100%)	1/3 (33%)
Mandible	21	20/21 (95%)	5/21 (24%)*
Total	24	23/24 (96%)	6/24 (25%)*

* Includes patient who tested positive to EPT but treated as if negative.

Table 27 Summary of Clinical Results during Endodontic Access in Teeth Negative to EPT/Green Endo-Ice after Supplemental Intraosseous Injection

Arch	No. of Teeth Negative to both EPT/Endo-Ice	Pain upon Entering Dentin	No. Requiring Second Intraosseous Inj. due to pain in dentin
Maxilla	3	0/3 (0%)	0 (0%)
Mandible	20	1/20 (5%)	1 (5%)
Total	23	1/23 (4%)	1 (4%)

Table 28 Summary of Clinical Results upon Pulp Exposure, Pulp Removal, or Filing in Teeth Negative to EPT/Green Endo-Ice after Supplemental IOI Injection

Arch	No. of Teeth Negative to both EPT/Endo-Ice	Pain upon Pulp Exposure	Pain upon Removing Pulp	Pain on Filing	Total No. with Pain	No. Requiring Intrapulpal Inj. due to Pulpal Pain	No. Requiring Second IOI Inj. due to Pulpal Pain*
Maxilla	3	0 (0%)	1 (33%)	0 (0%)	1 (33%)	1 (33%)	0/3 (0%)
Mandible	20	4 (20%)	0 (0%)	0 (0%)	4 (20%)	4 (20%)	1/21 (5%)*
Total	23	4 (17%)	1 (4%)	0 (0%)	6 (26%)	5 (22%)	1/23 (4%)*

* This includes patient from Table 24 who tested positive to EPT but was treated as negative, and reported pain upon pulpal exposure.

Table 29 Summary of Teeth Requiring Further Supplemental Anesthesia after Supplemental IOI Injection

Arch	Total No. of Teeth	No. Requiring Second IOI Inj. prior to access	No. Requiring Second IOI Inj. during Tx.	Total No. Requiring Second IOI Inj.	Total No. Requiring Intrapulpal Inj.	Total No. Requiring Second IOI and/or Intrapulpal Inj.
Maxilla	3	0/3 (0%)	0/3 (0%)	0/3 (0%)	1/3 (33%)	1/3 (33%)
Mandible	21	0/21 (0%)	2/21 (9%)	2/21 (9%)	3/21 (14%)	5/21 (24%)*
Total	24	0/24 (0%)	2/24 (8%)	2/24 (8%)	4/24 (17%)	6/24 (25%)*

* This total includes two patients in which the intraosseous injections were considered a success.

Table 30 Summary of Subject Response of Pain Ratings During Access/Endodontic Treatment Following Intraosseous Injection

Location	Number	Mild	Moderate	Severe
Pain entering dentin	1 (17%)	0 (0%)	1 (17%)	0 (0%)
Pain entering pulp	3 (50%)	2 (33%)	1 (17%)	0 (0%)
Pain removing pulp	2 (33%)	0 (0%)	2 (33%)	0 (0%)
Pain on filing	0 (0%)	0 (0%)	0 (0%)	0 (0%)

N=6

Table 31 Summary of Pulp Test Results in Teeth Positive to EPT, Green Endo-Ice, or both after Second Intraosseous Injection

Arch	Total No. of Pt.'s and Percentages	Positive Response To:				No. Requiring Intrapulpal Inj.
		EPT alone	Endo-Ice alone	Both Tests	Total	
Maxilla	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Mandible	2 (100%)	1 (50%)	0 (0%)	0 (0%)	1 (50%)	0 (0%)*
Total	2 (100%)	1 (50%)	0 (0%)	0 (0%)	1 (50%)	0 (0%)*

* Patient received treatment despite results of pulp tests.

Table 32 Summary of Clinical Results in Teeth Testing Negative to EPT and Green Endo-Ice after Second Intraosseous Injection

Arch	No. of Teeth Negative to both EPT/Endo-Ice	Clinical Results during Access					No. Requiring Intrapulpal Inj.
		pain entering dentin	pain entering pulp	pain removing pulp	pain on filing	no pain with tx.	
Maxilla	0/0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Mandible	1/2 (50%)	0 (0%)	1 (50%)*	0 (0%)	0 (0%)	1 (100%	1/2 (50%)*
Total	1/2 (50%)	0 (0)%	0 (0%)	0 (0%)	0 (0%)	1 (100%	1/2 (50%)*

* Includes patient from Table 26 who tested positive to EPT

Table 33 Summary of Time when Subject Responded with Pain Post-Intraosseous Injection (IOI Failures)

Patient #	Time Post-IOI Injection	Type of Secondary Supplemental Injection	Pain Rating of Intrapulpal Injection
4*	6:45	Intrapulpal	none
5	3:52	Second IOI	not required
7	9:27	Intrapulpal	mild
8	4:06	Second IOI	
8**	3:25	Intrapulpal	moderate
25*	12:30	Intrapulpal	none
36	4:33	Intrapulpal	mild
Mean	5:04		

* These patients were considered an intraosseous injection success and were not calculated into the mean time.

** This patient required an intrapulpal injection due to failure of the second intraosseous injection.

Table 34 Summary of Success/Failure of Intraosseous Injection

Arch	Number	Success	Failure
Maxilla	3	2 (66.67%)	1 (33.33%)
Mandible	21	19 (90.48%)	2 (9.52%)
Total	24	21 (87.5%)	3 (12.5%)

Fisher Exact Test = 0.343

95% Confidence Interval for Mandibular Success: 69.81% to 98.82%
99% Confidence Interval for Mandibular Success: 63.14% to 99.50%

APPENDIX A

BIOGRAPHICAL DATA

PATIENT INFORMATION								
patient information				*pre-study data*				
					Vitality Tests (EPT/Endolce)			Pain Rating of Tests
Subject	Sex	Age	Tooth #	Pain Rating	mesial	test	distal	EPT/Endolce
1	M	63	32	3	48/+	24/+	NA	
2	M	33	30	2	22/+	13/+	34/+	
3	M	23	3	3	52/-	49/+	80/-	
4	F	37	18	3	36/+	52/+	NA	
5	F	19	18	2	72/+	68/+	NA	
6	M	31	4	2	33/+	26/-	22/-	
7	M	29	14	2	30/+	16/+	NA	
8	F	39	19	1	40/-	39/+	37/-	
9	M	30	29	3	42/-	40/+	39/+	
10	F	40	14	2	27/+	67/+	80/+	
11	F	45	13	3	15/-	80/+	NA	
12	M	49	3	3	49/+	50/+	49/+	
13	F	40	14	1	NA	37/+	42/+	
14	F	29	20	3	27/+	45/+	22/+	
15	F	39	4	3	21/-	32/+	NA	
16	F	30	31	3	10/+	36/+	NA	
17	M	45	31	2	32/+	37/+	NA	
18	F	30	18	3	29/-	01/+	NA	
19	F	40	3	2	13/+	12/+	18/+	
20	F	21	19	3	27/+	52/+	80/+	
21	M	46	3	2	32/+	42/+	46/+	
22	F	28	4	3	62/+	32/+	48/+	
23	M	31	19	2	32/+	46/+	NA	
24	M	21	19	3	37/+	46/+	58/+	
25	F	54	31	1	NA	29/+	NA	
26	F	21	19	1	44/+	34/+	51/+	
27	F	27	19	2	36/+	45/+	49/+	
28	F	33	13	2	NA	35/+	46/+	
29	F	30	31	2	35/+	48/+	NA	
30	F	53	30	2	23/+	26/+	NA	
31	F	29	31	1	40/+	29/+	NA	
32	F	20	15	3	41/+	50/+	NA	
33	F	39	13	3	51/+	56/+	NA	
34	M	37	4	3	NA	42/+	80/-	
35	M	19	14	2	39/+	30/+	45/+	
36	M	24	18	3	NA	24/+	NA	
37	M	24	30	2	39/+	55/+	24/+	
38	F	30	30	2	40/+	53/+	53/+	3/3
39	F	63	5	2	20/+	37/+	37/+	2/2
40	M	53	31	1	NA/+	44/+	NA	1/3
41	M	19	3	3	30/+	49/-	49/-	1/NA
42	M	22	14	1	28/+	28/+	80/+	1/3
43	M	28	4	2	27/+	29/+	42/+	1/3
44	F	68	30	3	NA	68/+	NA	1/3
45	M	34	3	1	27/+	35/+	45/+	1/3
46	M	37	19	3	24/+	31/+	30/+	2/3
47	M	21	18	3	42/+	37/+	52/+	1/3
48	F	29	15	3	48/+	52/+	68/+	1/3
49	F	28	4	3	36/+	32/+	NA	3/3
50	F	24	4	3	28/+	36/+	45/+	1/3
51	M	32	3	2	NA	38/+	46/+	1/2

APPENDIX B

INFERIOR ALVEOLAR NERVE BLOCK/MAXILLARY INFILTRATION

RAW DATA

INFERIOR ALVEOLAR NERVE/INFILTRATION INJECTION DATA								
		pain ratings (0-3) of injection			*vitality tests* Minute 1			
Subject	Inj. Type	insertion	placement	deposition	EPT/Pain Rating	Endoice/Pain Rating	Lip Numb	Tongue Numb
1	IAN	0	0	0	42/	NO	NO	YES
2	IAN	0	1	1	40/	YES/	YES	YES
3	INF	0	1	0	80/	NO	NA	NA
4	IAN	1	0	1	56/	YES/	NO	YES
5	IAN	0	0	0	70/	NO	NO	NO
6	INF	0	1	1	80	NO	NA	NA
7	INF	1	1	2	23/	NO	NA	NA
8	IAN	1	0	0	24/	YES/	NO	YES
9	IAN	1	1	2	39/	YES/	YES	YES
10	INF	2	0	0	80	NO	NA	NA
11	INF	1	1	0	80	YES/	NA	NA
12	INF	0	0	1	54/	NO	NA	NA
13	INF	1	1	1	80	NO	NA	NA
14	IAN	2	2	1	55/	YES/	NO	YES
15	INF	0	0	0	60/	NO	NA	NA
16	IAN	2	0	0	80	NO	NO	YES
17	IAN	0	2	0	22/	YES/	YES	YES
18	IAN	1	0	0	03/	YES/	NO	NO
19	INF	1	1	2	23/	YES/	NA	NA
20	IAN	2	3	1	26/	YES/	NO	YES
21	INF	0	0	0	66/	NO	NA	NA
22	INF	1	0	0	80	NO	NA	NA
23	IAN	1	1	0	62/	YES/	NO	NO
24	IAN	1	2	2	55/	NO	YES	YES
25	IAN	1	2	2	80	NO	YES	YES
26	IAN	1	3	3	36/	YES/	YES	YES
27	IAN	2	3	2	25/	YES/	YES	YES
28	INF	1	1	1	80	NO	NA	NA
29	IAN	1	3	3	33/	YES/	NO	YES
30	IAN	1	0	2	55/	YES/	YES	YES
31	IAN	1	1	0	46/	NO	YES	YES
32	INF	0	0	1	47/	YES/	NA	NA
33	INF	0	1	1	80	NO	NA	NA
34	INF	1	1	0	41/	YES/	NA	NA
35	INF	0	0	0	80	NO	NA	NA
36	IAN	0	1	1	26/	YES/	YES	NO
37	IAN	2	2	2	35/1	YES/2	NO	YES
38	IAN	1	1	1	42/2	NO	YES	YES
39	INF	2	0	0	80	NO	NA	NA
40	IAN	1	1	1	35/1	NO	YES	YES
41	INF	1	1	0	46/1	NO	NA	NA
42	INF	1	0	0	45/1	NO	NA	NA
43	INF	0	0	0	80	NO	NA	NA
44	IAN	1	0	0	72/1	YES/2	YES	YES
45	INF	1	0	0	80	NO	NA	NA
46	IAN	1	1	0	29/2	YES/3	NO	NO
47	IAN	2	1	0	38/1	YES/3	NO	NO
48	INF	1	0	0	80	NO	NA	NA
49	INF	1	0	0	68/1	NO	NA	NA
50	INF	0	0	0	80	NO	NA	NA
51	INF	0	0	0	80	NO	NA	NA

INFERIOR ALVEOLAR NERVE/INFILTRATION INJECTION								
	vitality test continued							
	Minute 2				Minute 3			
Subject	EPT/Pain Rating	Endolce/Pain Rating	Lip Numb	Tongue Numb	EPT/Pain Rating	Endolce/Pain Rating	Lip Numb	Tongue Numb
1	80	NO	YES	YES	72/	NO	YES	YES
2	35/	YES/	YES	YES	28/	YES/	YES	YES
3	80	NO	NA	NA	80	NO	NA	NA
4	52/	YES/	NO	YEA	43/	YES/	NO	YES
5	80	YES/	NO	YES	80	YES/	NO	YES
6	80	NO	NA	NA	80	NO	NA	NA
7	40/	NO	NA	NA	80	NO	NA	NA
8	25/	YES/	NO	YES	26/	NO	NO	YES
9	80	YES/	YES	YES	80	NO	YES	YES
10	80	NO	NA	NA	80	NO	NA	NA
11	80	NO	NA	NA	80	NO	NA	NA
12	80	NO	NA	NA	80	NO	NA	NA
13	80	NO	NA	NA	80	NO	NA	NA
14	44/	YES/	NO	YES	57/	YES/	NO	YES
15	80	NO	NA	NA	80	NO	NA	NA
16	43/	NO	YES	YES	80	NO	YES	YES
17	39/	YES/	YES	YES	50/	NO	YES	YES
18	23/	YES/	NO	NO	23/	YES/	NO	NO
19	41/	NO	NA	NA	80	NO	NA	NA
20	62/	YES/	NO	YES	34/	YES/	NO	YES
21	80	NO	NA	NA	80	NO	NA	NA
22	80	NO	NA	NA	80	NO	NA	NA
23	80	NO	YES	YES	80	NO	YES	YES
24	80	NO	YES	YES	80	NO	YES	YES
25	41/	NO	YES	YES	43/	NO	YES	YES
26	33/	YES/	YES	YES	39/	NO	YES	YES
27	21/	YES/	YES	YES	26/	YES/	YES	YES
28	80	NO	NA	NA	80	NO	NA	NA
29	39/	YES/	NO	YES	36/	NO	YES	YES
30	17/	NO	YES	YES	26/	NO	YES	YES
31	46	NO	YES	YES	55/	NO	YES	YES
32	42/	YES/	NA	NA	41/	YES/	NA	NA
33	80	NO	NA	NA	80	NO	NA	NA
34	40/	YES/	NA	NA	44/	YES/	NA	NA
35	80	NO	NA	NA	80	NO	NA	NA
36	33/	NO	YES	YES	37/	NO	YES	YES
37	38/1	NO	NO	YES	62/1	YES/2	NO	YES
38	67/1	NO	YES	YES	70/1	NO	YES	YES
39	80	NO	NA	NA	80	NO	NA	NA
40	67/1	NO	YES	YES	67/1	NO	YES	YES
41	80	NO	NA	NA	80	NO	NA	NA
42	60/1	NO	NA	NA	80	NO	NA	NA
43	80	NO	NA	NA	80	NO	NA	NA
44	80	NO	YES	YES	80	NO	YES	YES
45	80	NO	NA	NA	80	NO	NA	NA
46	32/2	YES/3	NO	YES	27/2	YES/3	NO	YES
47	40/1	YES/3	NO	YES	46/1	YES/3	NO	YES
48	80	NO	NA	NA	80	NO	NA	NA
49	80	NO	NA	NA	80	NO	NA	NA
50	80	NO	NA	NA	80	NO	NA	NA
51	80	NO	NA	NA	80	NO	NA	NA

INFERIOR ALVEOLAR NERVE/INFILTRATION INJECTION DATA								
	vitality tests continued							
	Minute 4				Minute 5			
Subject	EPT/ Pain Rating	Endolce/Pain Rating	Lip Numb	Tongue Numb	EPT/Pain Rating	Endolce/Pain Rating	Lip Numb	Tongue Numb
1	56/	NO	YES	YES	80	NO	YES	YES
2	34/	NO	YES	YES	39	NO	YES	YES
3								
4	80	YES/	NO	YES	50/	YES	NO	YES
5	80	NO	YES	YES	54/	NO	YES	YES
6								
7								
8	33/	YES/	NO	YES	31/	YES/	NO	YES
9	80	NO	YES	YES	80	NO	YES	YES
10								
11								
12								
13								
14	57/	YES/	YES	YES	59/	YES/	YES	YES
15								
16	80	NO	YES	YES	80	NO	YES	YES
17	36/	NO	YES	YES	36/	NO	YES	YES
18	26/	YES/	YES	NO	25/	YES/	YES	NO
19								
20	39/	YES	NO	YES	42/	YES/	NO	YES
21								
22								
23	80	NO	YES	YES	80	NO	YES	YES
24	80	NO	YES	YES	80	NO	YES	YES
25	80	NO	YES	YES	47/	NO	YES	YES
26	49/	NO	YES	YES	42/	NO	YES	YES
27	48/	NO	YES	YES	80	YES/	YES	YES
28								
29	35/	NO	YES	YES	35/	NO	YES	YES
30	32/	NO	YES	YES	32/	NO	YES	YES
31	51/	NO	YES	YES	52/	NO	YES	YES
32								
33								
34								
35								
36	47/	NO	YES	YES	44/	NO	YES	YES
37	58/1	YES/2	NO	YES	55/1	YES/2	NO	YES
38	80	NO	YES	YES	80	NO	YES	YES
39								
40	68/1	NO	YES	YES	80	NO	YES	YES
41								
42								
43								
44	80	NO	YES	YES	80	NO	YES	YES
45								
46	29/2	YES/3	NO	YES	36/2	YES/3	NO	YES
47	43/1	YES/3	NO	YES	44/1	YES/3	NO	YES
48								
49								
50								
51								

INFERIOR ALVEOLAR NERVE/INFILTRATION INJECTION DATA									
		vitality tests second IAN							
	2nd IAN	Minute 1				Minute 2			
Subject	Required	EPT/Pain Rating	Endoice/Pain Rating	Lip Numb	Tongue Numb	EPT/Pain Rating	Endoice/Pain Rating	Lip Numb	Tongue Numb
1	NO								
2	NO								
3	NA								
4	YES	64/	NO	YES	YES	80	NO	YES	YES
5	NO								
6	NA								
7	NA								
8	YES	80	NO	YES	YES	56/	NO	YES	YES
9	NO								
10	NA								
11	NA								
12	NA								
13	NA								
14	NO								
15	NA								
16	NO								
17	NO								
18	YES	20/	YES/	YES	YES	24/	YES/	YES	YES
19	NA								
20	YES	55/	YES/	NO	YES	67/	YES/	NO	YES
21	NA								
22	NA								
23	NO								
24	NO								
25	NO								
26	NO								
27	NO								
28	NA								
29	NO								
30	NO								
31	NO								
32	NA								
33	NA								
34	NA								
35	NA								
36	NO								
37	YES	80	NO	YES	YES	80	NO	YES	YES
38	NO								
39	NA								
40	NO								
41	NA								
42	NA								
43	NA								
44	NO								
45	NA								
46	YES	80	YES/3	YES	YES	80	YES/1	YES	YES
47	YES	46/1	YES/3	YES	YES	41/1	YES/3	YES	YES
48	NA								
49	NA								
50	NA								
51	NA								

INFERIOR ALVEOLAR NERVE/ INFILTRATION INJECTION DATA								
	vitality tests second IAN							
	Minute 3				Minute 4			
Subject	EPT/Pain Rating	Endolce/Pain Rating	Lip Numb	Tongue Numb	EPT/Pain Rating	Endolce/Pain Rating	Lip Numb	Tongue Numb
1								
2								
3								
4	80	NO	YES	YES	80	NO	YES	YES
5								
6								
7								
8	52/	NO	YES	YES	59/	NO	YES	YES
9								
10								
11								
12								
13								
14								
15								
16								
17								
18	21/	YES/	YES	YES	34/	YES/	YES	YES
19								
20	57/	YES/	NO	YES	58/	YES/	YES	YES
21								
22								
23								
24								
25								
26								
27								
28								
29								
30								
31								
32								
33								
34								
35								
36								
37	80	NO	YES	YES	80	NO	YES	YES
38								
39								
40								
41								
42								
43								
44								
45								
46	80	YES/1	YES	YES	80	YES/1	YES	YES
47	42/1	YES/1	YES	YES	48/1	YES/1	YES	YES
48								
49								
50								
51								

INFERIOR ALVEOLAR NERVE/INFILTRATION INJECTION DATA							
	vitality tests continued Minute 5				Is Supplemental Anesthesia Required?		
Subject	EPT/Pain Rating	Endolce/Pain Rating	Lip Numb	Tongue Numb	Y/N	Reason/Pain Rating	Type/Pain Rating
1					YES	Pain Enter Dentin/2	IOI
2					YES	Positive EPT	IOI
3					NO		
4	80	NO	YES	YES	YES	Pain Enter Dentin/2	IOI
5					YES	Pain Enter Dentin/2	IOI
6					YES	Pain Enter Pulp/1	Intrapulpal/1
7					YES	Pain Enter Dentin/2	IOI
8	59/	NO	YES	YES	YES	Positive EPT	IOI
9					YES	Pain Remove Pulp/3	Intrapulpal/2
10					NO		
11					NO		
12					YES	Filing/1	Intrapulpal/0
13					NO		
14					YES	Positive EPT/Endolce	IOI
15					NO		
16					YES	Pain Enter Pulp/3	IOI
17					YES	Positive EPT	IOI
18	25/	NO	YES	YES	YES	Positive EPT	IOI
19					NO		
20	58/	YES/	YES	YES	YES	Positive EPT/Endolce	IOI
21					NO		
22					NO		
23					YES	Pain Enter Dentin/1	IOI
24					YES	Pain Remove Pulp//2	Intrapulpal/1
25					YES	Positive EPT	IOI
26					YES	Positive EPT	IOI
27					YES	Positive Endolce	IOI
28					NO		
29					YES	Positive EPT	IOI
30					YES	Positive EPT	IOI
31					YES	Positive EPT	IOI
32					YES	Positive EPT/Endolce	IOI
33					NO		
34					YES	Positive EPT/Endolce	IOI
35					NO		
36					YES	Positive EPT	IOI
37	80	NO	YES	YES	YES	Pain Enter Dentin/2	IOI
38					NO		
39					NO		
40					YES	Pain Enter Pulp/2	Intrapulpal/2
41					YES	Pain Remove Pulp/1	Intrapulpal/1
42					YES	Pain Remove Pulp/2	Intrapulpal/1
43					NO		
44					NO		
45					NO		
46	80	YES/1	YES	YES	YES	Positive Endolce	IOI
47	48/1	YES/1	YES	YES	YES	Positive EPT/Endolce	IOI
48					NO		
49					NO		
50					NO		
51					YES	Pain Remove Pulp/2	Intrapulpal/2

APPENDIX C

INTRAOSSEOUS INJECTION RAW DATA

INTRAOSSEOUS INJECTION DATA						
	pain ratings of injection (0-3)					
	Infiltration		Penetration		Subjective	Back-Pressure
Subject	insertion	deposition	perforation	injection	Cardiac Response	on Injection
1	2	0	0	3	YES	NO
2	1	0	0	0	NO	NO
3						
4	1	0	0	1	YES	NO
5	2	0	0	0	NO	NO
6						
7	0	0	3	1	YES	NO
8	0	0	0	0	NO	NO
9						
10						
11						
12						
13						
14	1	0	0	0	YES	YES
15						
16	1	2	0	1	YES	NO
17	1	0	1	0	NO	NO
18	1	0	0	0	NO	NO
19						
20	1	0	1	0	NO	NO
21						
22						
23	0	0	1	0	YES	NO
24						
25	0	0	0	0	NO	NO
26	3	3	0	0	NO	NO
27	1	0	0	0	YES	NO
28						
29	0	0	0	1	YES	NO
30	0	0	0	0	YES	YES
31	0	0	1	0	YES	NO
32	1	0	1	0	NO	NO
33						
34	0	0	2	0	YES	YES
35						
36	0	0	0	0	NO	NO
37	1	0	0	1	NO	NO
38						
39						
40						
41						
42						
43						
44						
45						
46	0	1	1	0	NO	NO
47	1	0	0	0	NO	NO
48						
49						
50						
51						

INTRAOSSEOUS INJECTION DATA						
	vitality tests					
	Minute 1		Minute 2		Minute 3	
Subject	EPT/Pain Rating	Endolce/Pain Rating	EPT/Pain Rating	Endolce/Pain Rating	EPT/Pain Rating	Endolce/Pain Rating
1	80	NO	80	NO	80	NO
2	80	NO	66/	NO	80	NO
3						
4	80	NO	80	NO	80	NO
5	80	NO	80	NO	80	NO
6						
7	80	NO	80	NO	80	NO
8	52/	N0	58/	NO	58/	NO
9						
10						
11						
12						
13						
14	80	NO	80	NO	80	NO
15						
16	80	NO	80	NO	80	NO
17	80	NO	80	NO	80	NO
18	80	NO	80	NO	80	NO
19						
20	80	NO	80	NO	80	NO
21						
22						
23	80	NO	80	NO	80	NO
24						
25	80	NO	80	NO	80	NO
26	80	NO	80	NO	80	NO
27	80	NO	80	NO	80	NO
28						
29	80	NO	80	NO	80	NO
30	80	NO	80	NO	80	NO
31	80	NO	80	NO	80	NO
32	80	NO	80	NO	80	NO
33						
34	80	NO	80	NO	80	NO
35						
36	80	NO	80	NO	80	NO
37	80	NO	80	NO	80	NO
38						
39						
40						
41						
42						
43						
44						
45						
46	80	NO	80	NO	80	NO
47	62/1	YES/1	80	NO	80	NO
48						
49						
50						
51						

INTRAOSSEOUS INJECTION DATA				
	Is Supplemental Anesthesia Required?			Time Pain Occurs Post-IOI Injection
Subject	Y/N	Reason/Pain Rating	Type/Pain Rating	(minutes)
1	NO			
2	NO			
3				
4	YES	Pain Enter Pulp/1	Intrapulpal/0	6:45
5	YES	Pain Enter Dentin/2	Second IOI	3:52
6				
7	YES	Pain Pemove Pulp/2	Intrapulpal/1	9:27
8	YES	Pain Enter Pulp/2	Second IOI	4:06
9				
10				
11				
12				
13				
14	NO			
15				
16	NO			
17	NO			
18	NO			
19				
20	NO			
21				
22				
23	NO			
24				
25	YES	Pain Remove Pulp/2	Intrapulpal/0	11:30
26	NO			
27	NO			
28				
29	NO			
30	NO			
31	NO			
32	NO			
33				
34	NO			
35				
36	YES	Pain Enter Pulp/1	Intrapulpal/1	4:32
37	NO			
38				
39				
40				
41				
42				
43				
44				
45				
46	NO			
47	NO			
48				
49				
50				
51				

INTRAOSSEOUS INJECTION DATA						
	pain ratings second injection (0-3)					
	Infiltration		Penetration		Subjective	Back-Pressure
Subject	insertion	deposition	perforation	injection	Cardiac Response	on Injection
1						
2						
3						
4						
5	0	0	0	0	YES	NO
6						
7						
8	0	0	2	0	NO	NO
9						
10						
11						
12						
13						
14						
15						
16						
17						
18						
19						
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51						

INTRAOSSEOUS INJECTION DATA						
	2nd intraosseous injection vitality tests					
	Minute 1		Minute 2		Minute 3	
Subject	EPT/Pain Rating	EndoIce/Pain Rating	EPT/Pain Rating	EndoIce/Pain Rating	EPT/Pain Rating	EndoIce/Pain Rating
1						
2						
3						
4						
5	80	NO	80	NO	80	NO
6						
7						
8	58/	NO	55/	NO	52/	NO
9						
10						
11						
12						
13						
14						
15						
16						
17						
18						
19						
20						
21						
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48						
49						
50						
51						

INTRAOSSEOUS INJECTION DATA					
	Is Supplemental Anesthesia Required?			Time Pain Occurs Post-IOI Injection	INTRAOSSEOUS INJECTION SUCCESS or FAILURE
Subject	Y/N	Reason/Pain Rating	Type/Pain Rating	(minutes)	
1					SUCCESS
2					SUCCESS
3					NA
4					SUCCESS
5	NO				SUCCESS
6					NA
7					FAILURE
8	YES	Pain Enter Pulp/2	Intrapulpal/2	3:25	FAILURE
9					NA
10					NA
11					NA
12					NA
13					NA
14					SUCCESS
15					NA
16					SUCCESS
17					SUCCESS
18					SUCCESS
19					NA
20					SUCCESS
21					NA
22					NA
23					SUCCESS
24					NA
25					SUCCESS
26					SUCCESS
27					SUCCESS
28					NA
29					SUCCESS
30					SUCCESS
31					SUCCESS
32					SUCCESS
33					NA
34					SUCCESS
35					NA
36					FAILURE
37					SUCCESS
38					NA
39					NA
40					NA
41					NA
42					NA
43					NA
44					NA
45					NA
46					SUCCESS
47					SUCCESS
48					NA
49					NA
50					NA
51					NA

APPENDIX D

DATA RECORDING SHEET

DATA RECORDING SHEET FOR THE ANESTHETIC EFFICACY OF THE INTRAOSSEOUS INJECTION IN IRREVERSIBLE PULPITIS

Pt. Number _____ Tooth # _____

Pre-Study Tests:

	M Tooth	Test Tooth	D Tooth
EPT			
EndoIce			

Pain Rating of Test Tooth (0-3):_____
0=none, 1=mild, 2=moderate, 3=severe

Post-IAN/Inf Tests:

Minute	EPT	ENDOICE(+/-)	Lip Numb	Tongue Numb
1				
2				
3				
4				
5				

Pain Ratings - (0-3):
IAN insertion: _____ Inf. insertion: _____
placement: _____ placement: _____
deposition:_____ deposition:_____

Is a second IAN required? Y or N

Post-IOI Tests:

Pain Rating of Injection (0-3):
Infiltration; insertion: _____ deposition: _____
Penetration; perforation: _____ deposition: _____
Subjective Cardiac Response Y or N
Back-Pressure on Injection Y or N

Minute	EPT	ENDOICE
1		
2		
3		

Second IOI Tests:

Pain Rating of Injection (0-3):
Infiltration; insertion _____ deposition _____
Penetration; perforation _____ deposition _____
Subjective Cardiac Response Y or N
Back-Pressure on Injection Y or N

Minute	EPT	ENDOICE
1		
2		
3		

Pain Experience During Access.

None Entering Dentin Entering Pulp Removing Pulp (placing files to length) Filing

Time Pain Occurs (from stopwatch)_____

Supplemental Injections Required? Y or N
Type: _____ IOI Success/Failure

APPENDIX E

CONSENT FORM

THE OHIO STATE UNIVERSITY **Protocol No._____**

CONSENT TO INVESTIGATIONAL TREATMENT OR PROCEDURE

I,_____, hereby authorize or direct Dr. John M. Nusstein associates or assistants of his choosing, to perform the following treatment or procedure (describe in general terms).

I have a tooth which is hurting (painful) and I am aware that it either needs root canal treatment or it needs to be extracted. The tooth causing me pain will be tested with an electric pulp tester and an ice spray and I will rate the pain I am having prior to any treatment. My tooth will experience a tingling sensation or slight discomfort (pain) when electric pulp tested, at which time the device will be removed immediately. Testing with ice may cause the tooth to hurt (pain). One injection (shot) will be given for lower teeth in the back of my jaw (the inferior alveolar injection) using 2% lidocaine with 1:100,000 epinephrine (a numbing solution like novocaine). If my lip does not get numb, I will get another injection (shot) in the back of my jaw. Upper teeth will get two injections (buccal infiltration) of the same anesthetic solution right next to the tooth. The electric pulp tester and ice will then be used every other minute for up to 5 minutes to test the numbness (no pain) of my tooth. If my tooth is not numb, I will get another injection behind my tooth, in the gums (the intraosseous injection). The intraosseous injection consists of drilling a very small hole through my gums and bone; I will then be given an injection (shot) of anesthetic solution through this hole. The tooth will then be tested every minute for 3 minutes for numbness using ice and the electric pulp tester. I will be asked to rate the pain I feel when the teeth are tested, and while the injections (shots) are being given to me. If my tooth is numb, emergency root canal treatment will be started. If my tooth is not numb, other injections, such as: giving another intraosseous injection either in the same area (back of the tooth) or in front of the tooth; or an intrapulpal injection (shot) directly into the pulp (inside) of my tooth, will be given to get my tooth numb. I realize that if I am pregnant or suspect a pregnancy, I cannot participate in the study. My participation or non-participation will have no effect on whether I will receive emergency treatment.

upon _____
(myself or name of subject)

The experimental (research) portion of the treatment or procedure is:

I will get a local anesthetic injection(s) (shot) to try and get the tooth that is bothering me numb. The tooth will then be tested with ice and an electric pulp tester to determine if the tooth is completely numb. If it is not numb, I will receive an intraosseous injection (shot) behind my tooth and it will be tested again with ice and an electric pulp tester. If it is still

not numb, another intraosseous injection (shot) may be given in back of my tooth, or in front of it. Once the tooth is numb, the root canal treatment will be started. **This is done as part of an investigation entitled:**

The Anesthetic Efficacy of the Intraosseous Injection in Irreversible Pulpitis.

1. Purpose of the procedure or treatment: To determine whether giving an intraosseous injection next to an inflamed tooth (tooth with a toothache), after regular injections (an inferior alveolar, or buccal infiltration injection) will provide pulpal anesthesia (numbness) for endodontic (root canal) treatment.

2. Possible appropriate alternative procedures to treatment (not to participate in the study is always an option): To have my tooth treated endodontically (root canal treatment) using other injection techniques to achieve numbness or have it removed without being in this study. I may choose not to participate in the study, or if I participate, I may withdraw from the study at any time.

3. Discomforts and risks reasonably to be expected: I am aware that I may have pain associated with the local anesthetic (numbing solution) and/or intraosseous injection (shot) or soreness at the site of the injection (shot) for approximately two days. Where I receive the injection, I may have swelling (hematoma - a collection of blood under the skin) or a bruise may develop. I may experience a feeling of anxiety or my heart may pound faster. I may also experience lightheadedness or fainting. My toothache pain may stay the same or get worse during the study. The tingling sensation and/or slight discomfort (pain) produced by the pulp tester and ice may be uncomfortable to me. I may have a possible allergic reaction to the local anesthetic (itching or hives) or an unexpected infection (rare) which could result in permanent nerve damage. There may be soreness of my gum tissue for a few days or a possible altered sensation of my lip or tongue which may last up to a few weeks.

4. Possible benefits for subjects/society: I will not directly benefit from this study other than the free emergency treatment for my toothache, which normally costs $50.00. Society may ultimately benefit from the insight that we can or cannot achieve painless root canal treatment after giving an intraosseous injection next to a painful tooth.

5. Anticipated duration of the subject's participation (including number of visits): I am aware that I may have to delay emergency treatment of my tooth until I get numb. I will then have emergency treatment started. The visit will last approximately 90 minutes.

I hereby acknowledge that Dr. John M. Nusstein has provided information about the procedure described above, about my rights as a subject, and he answered all questions to my satisfaction. I understand that I may contact him at phone number 292-5399 should I have additional questions. He has explained the risks described above and I understand them; he also offered to explain all possible risks or complications.

I understand that, where appropriate, the U.S. Food and Drug Administration may inspect records pertaining to this study. I understand further that records obtained during my participation in this study that may contain my name or other personal identifiers may be made available to the sponsor of this study. Beyond this, I understand that my participation will remain confidential.

I understand that I am free to withdraw my consent and participation in this project at any time notifying the project director without prejudicing future care. No guarantee has been given to me concerning this treatment or procedure.

I understand in signing this form that, beyond giving consent, I am not waiving any legal rights that I might otherwise have, and I am not releasing the investigator, the sponsor, the institution, or its agents from any legal liability for damages that they might otherwise have.

In the event of injury resulting from participation in this study, I also understand that immediate medical treatment is available at University Hospitals of The Ohio State University and that the costs of such treatment will be at my expense; financial compensation beyond that required by law is not available. Questions about this should be directed to the Office of Research Risks at 292-5958.

I have read and fully understand the consent form. I sign it freely and voluntarily. A copy has been given to me.

Date:_____ Time:_____ AM/PM Signed_____
(subject)

Witness(es) (If Required) _____

_____

_____
Person Authorized to Consent for Subject if Required

I certify that I have personally completed all blanks for this form and explained them to the subject or his/her representative before requiring the subject or his/her representative to sign it.

Date: _____ Signed: _____
(Signature of Project Director or his/her Authorized Representative)

APPENDIX F

HUMAN SUBJECTS REVIEW COMMITTEE

INFORMATION FORM

BIOMEDICAL SCIENCES

SUMMARY SHEETS

ADDRESS EACH ITEM IN A COMPLETE AND CONCISE MANNER. (Do not leave any item blank with "See attached.") Use continuation pages when necessary.

1. Abstract (overview of research).

The purpose of this study will be to determine whether a Stabident Intraosseous injection (IOI) of 2% lidocaine with 1:100,000 epinephrine used as a supplemental technique, after standard injections, will provide anesthesia in patients with painful inflamed pulps (toothache).

Eighty adult male and female subjects presenting for emergency treatment will be selected for this study. The patient must have a posterior tooth with a clinical diagnosis of irreversible pulpitis as determined by pulp tests using an electric pulp tester and EndoIce (dichlorodifluoromethane).

Tooth vitality will be tested using the EPT and EndoIce every other minute for 5 minutes after receiving the inferior alveolar, or buccal infiltration injection. After 5 minutes, failure to achieve an 80/80 reading and a negative response to the EndoIce will lead to the administration of the intraosseous injection. Testing will resume at 1 minute intervals until anesthesia is achieved within 3 minutes.

Teeth will be accessed in the standard manner and patients will be asked to respond if any pain is felt during the access procedure. The need for further anesthesia will be considered a failure in the IOI injection.

2. Describe the requirements for a subject population and explain the rationale for using in this population special groups such as prisoners, children, the mentally disabled or groups whose ability to give voluntary informed consent may be in question. Address means of pregnancy screening for females.

The subjects used in this study will be healthy adults who have a symptomatic posterior tooth with a clinical diagnosis of irreversible pulpitis. The subjects for this study will be chosen from the patients of record and emergency patients of The College of Dentistry, The Ohio State University. All subjects will give voluntary, informed consent prior to participation in this study. In no instance will a subject participate who is a prisoner, a

child, mentally retarded, or who is a member of any group whose ability to give voluntary, informed consent may be in question. All female subjects will be questioned regarding a suspected pregnancy and will complete a medical history and informed consent form prior to participation in this study.

3. Describe and assess any potential risks - physical psychological, social, legal, financial, or other - and assess the likelihood and seriousness of such risks. If methods of research create potential risks, describe other methods, if any, that were considered and why they will not be used.

Since conventional local anesthetic and Stabident intraosseous injection techniques will be used, the discomforts and risks would include: possible soreness of the injection site which dissipates in a few days, a short-lasting increase in heart rate, allergy to the anesthetic, syncope, a slight risk of infection, and possible tingling of the tongue, lip, and cheek that may persist for a few weeks, but is rare, hematoma or bruise may develop at the site of the injections.

4. Describe consent procedures to be followed, including how and where informed consent will be obtained. (The use of a finder's fee for recruiting subjects is not permitted.)

The possible risks involved will be explained to the subject's satisfaction prior to the patient reading and signing the consent form.

5. Describe procedures (including confidentiality safeguards) for protecting against or minimizing potential risks and an assessment of their likely effectiveness.

The identity of the subjects will be by random number and will be kept confidential to unauthorized personnel. Essential emergency medical equipment, drugs, and personnel with C.P.R. and A.C.L.S. training will be immediately available. The likelihood of a situation arising that cannot be handled by said personnel and equipment is extremely remote.

6. Assess the potential benefits to be gained by the individual subjects, as well as benefits which may accrue to society in general as a result of the planned work.

Subjects will receive emergency endodontic treatment, normally $50.00, at no charge.

Society may ultimately benefit if an anesthetic technique which is more effective than the one used today in symptomatic teeth is discovered.

7. Compare the risks versus the benefits.

The risks of the inferior alveolar and Stabident Intraosseous injections are minimal. The side effects and risks of the anesthetic administered is also minimal in the amounts to be used. Therefore, the risk-benefit ratio is favorable.

8. Will the subjects for the study be paid for participating in this study.

_X_ No ___ Yes--How much?

Will subjects be paid for selected activities (e.g., blood drawing) or for general participation in the study?

The subjects will receive emergency treatment for the offending tooth, normally $50.00, at no charge.

* NOTE: All information concerning payment, including the amount and schedule of payment, must be included in the consent form.

Is there any other inducement? If so, please describe.

_X_ No ___ Yes--Please describe.

9. Will advertising be used to recruit subjects? ** If yes, attach a copy of the proposed advertisement.

_X_ No ___ Yes

SOURCE OF FUNDING FOR PROPOSED RESEARCH: (Check A or B):

A. OSURF: Sponsor _____ RF Proposal/Project No._____

B. Other (Identify) Endodontic Support Fund

Information about the funding/sponsorship of human subjects research activities is required for administrative purposes. Such information is generally not required as part of the human subjects review process.

APPENDIX G

HEALTH HISTORY FORM

THE OHIO STATE UNIVERSITY
COLLEGE OF DENTISTRY

DENTAL-MEDICAL HISTORY

Name _____.

Medical History

(Please answer the questions by circling either NO or YES. If you are uncertain about the question, leave it unanswered. RESPOND ONLY to questions 1-25.)

Past Medical History

1. Have you had any serious illness or been hospitalized?	NO	YES
2. Have you had rheumatic fever or rheumatic heart disease?	NO	YES
3. Do you have or have you had a heart murmur (leaky valve) or mitral valve prolapse?	NO	YES
4. Have you had or do you have any heart or blood vessel disease such as a heart attack or stroke?	NO	YES
5. Have you been told that your blood pressure is too high?	NO	YES
6. Have you had jaundice (yellow skin) or hepatitis?	NO	YES
7. Have you been treated for a seizure disorder (convulsions or epilepsy)?	NO	YES
8. Have you had a tumor or disease that required x-ray, radium or cobalt treatments?	NO	YES
9. Have you had excessive or prolonged bleeding following a cut, tooth extraction, or other injury?	NO	YES
10. Have you had an allergic or unusual reaction to any drugs or medications (penicillin, codeine, aspirin, etc.)?	NO	YES
11. Do you have any allergies?	NO	YES
12. Are you currently taking any drugs or medications (such as anti-biotics, blood thinners, cortisone, tranquilizers, or heart medicine)?	NO	YES
13. Are you currently under the care of a physician (M.D., D.O.)?	NO	YES
14. Approximately how long has it been since you were last seen by a physician? _____		
15. Do you have any disease, condition, or problem not listed above?	NO	YES

Systems Review

16. Do you have a sore or hoarse throat?	NO	YES
17. Do you have a persistent cough or sometimes cough up blood?	NO	YES
18. Do you get pains in the heart or chest (angina pectoris)?	NO	YES
19. Does climbing one flight of stairs make you tired and require you to stop and rest so that you can catch your breath?	NO	YES

20. Are you pregnant or nursing at the present time? NO YES
    E.D.D._____Gravida_____Para_____

Dental History

21. Have you had any trouble associated with previous dental treatment (dizziness, fainting or reaction to novocain)? NO YES
22. Do you have any lumps or sores in your mouth now? NO YES
23. Do you feel you will eventually lose all your teeth? NO YES
24. Are you nervous about receiving any dental treatment? NO YES
25. Have you had regular dental checkups? NO YES
    When was your last dental visit? _____

Current Medications

Trade Name	Generic Name	Dose & Frequency	Reason

Medical Consultation Required? (No - Yes)
Date Consultation Form Sent _____

Summary of patient's medical status

_____

To the best of my knowledge, the above information is correct and complete.

_____ _____
Patient's Signature Instructor

_____ _____
Student Date

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